PEPTIC ULCERATION

A SYMPOSIUM FOR SURGEONS

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FOREWORD

THE problem of the cause of gastric and duodenal ulcer is a part of the more general question of the resistance of the gastrointestinal tract to the digestive action of its own secretions John Hunter interested himself in this problem as in so many others and explained the resistance of the normal gastric wall to digestion on the basis of a vital principle in living cells The familiar question why does not the stomach digest itself should probably be changed since under some conditions and in certain individuals the stomach does digest itself and the result is the so-called peptic ulcer It might be more profitable to add the question What are the abnormal conditions which make possible digestion of a portion of the gastric wall? Resistance to gastric digestion is however not a property specifically limited to the mucosa of the gastrointestinal tract Organs such as the spleen kidney and pancreas implanted into large defects produced in the stomach wall of dogs are not digested away by the usual gastric content of these animals Experiments have demonstrated however that the mucosa has greater resistance to peptic digestion than the living cells of the spleen or kidney and the mucosa of the stomach is more resistant than the mucosa of the oesophagus or small intestine

Virchow and Hauser advanced the idea that a local decrease in resistance of the mucosa could be produced by thrombosis or embolism of terminal arteries in the wall of the stomach or duodenum and the devitalised area digested by the gastric content In more modern times Konjetzny has pursued this side question believing that peptic ulcers can usually be accounted for on the basis of a decreased resistance of a localised area of the mucosa In this symposium on peptic ulcer Sircus has provided an excellent critique of this problem

The experimental demonstration that pure gastric juice as it is secreted by the fundic glands has the capacity to destroy and digest all living cells including those of the stomach and duodenum has helped to bring this question into focus The usual gastric content does not digest living cells probably because the acid and pepsin of the gastric juice are partially buffered and neutralised by swallowed food saliva and regurgitated intestinal secretions I do not know of a satisfactory explanation for the remarkable fact that the pure parietal secretion can digest the gastric wall as well as the gland cells that have produced the gastric juice However should conditions arise in patients where the mucous membrane is exposed for any long period of time to undiluted normal gastric juice a sufficient cause for peptic ulcer is present without postulating a defect in the mechanism of resistance A hypersecretion

of gastric juice particularly one brought about without the ingestion of food might be expected to produce such a situation It is thus not surprising that the excessive secretion of gastric juice in the empty stomach of patients with pancreatic tumours of the type described by Zollinger and Ellison produces progressive ulceration in all areas of mucosa exposed to it and without evident local pathology to cause a decrease in resistance It is my own conviction that hypersecretion of gastric juice is a sufficient cause for most chronic progressive ulcers This hypersecretion in duodenal ulcer patients is usually of nervous origin and is brought on by the tensions and strains of modern life Many gastric ulcers on the other hand are caused by a hypersecretion of gastric juice of humoral origin due to stasis of food in the stomach because of gastric atony or pyloric stenosis from a pre existent duodenal ulcer Prolonged contact of food with the gastric antrum appears to be an adequate stimulus for the continued release of gastrin and consequent prolonged stimulation of gastric secretion

The surgical treatment of peptic ulcer as outlined by Professor Wells has developed both from the concept that the lesion is due to local pathology in the wall of the stomach and duodenum and to the aggressive action of the gastric juice Konjetzny was interested in excising the antrum because he believed that the gastritis present in so many resected stomachs indicated abnormal mucosa that would subsequently break down and form an ulcer Perhaps the majority of surgeons resect the stomach in ulcer patients to decrease the quantity of gastric juice secreted While removal of the antrum excludes the source of gastrin this is not usually sufficient in patients with duodenal ulcer and consequently large portions of the fundus of the stomach have also been excised While extensive gastric resection has usually reduced the secretion of gastric juice sufficiently to prevent recurrent ulceration the loss of the storage function of the stomach has brought other evils in its train These have been admirably discussed by Professor Welbourn in his analysis of alimentary function after gastrectomy and the so-called post gastrectomy syndrome The translation into English of leading German articles on gastric surgery add to the value of this excellent symposium and it is a pleasure and privilege for me to provide this introductory statement

LESTER R DRAGSTEDT

1960

PREFACE

THIS symposium presents in considerable detail recently acquired knowledge and changes in practice within the more lightly sketched framework of what is generally agreed concerning gastric and duodenal ulceration No attempt has been made to describe in detail techniques which can only be mastered in the operating theatre but space is devoted to methods of dealing with unexpected difficulties which may arise in the course of everyday procedures Little is said about routine diagnosis and such like but exceptions from the general pattern are discussed at some length By contrast aetiological factors and the post gastrectomy syndromes are described very fully The content of the various sections is in fact proportional to the amount of new or little known material they contain rather than to their relative importance in day to day surgery Some basic knowledge is assumed

We feel we have some historical justification for presenting this symposium The first recorded Billroth I gastrectomy in Britain was done in the Royal Southern Hospital Liverpool Twenty years ago Ronald Edwards did the first reported experimental work on vagotomy applied to duodenal ulcer in this Department where later R B Welbourn made the observations which form so large a part of the foundations of our knowledge of the post gastrectomy syndromes

Our team with one exception is composed of individuals in of or lately at this School We are grateful to them for their contributions in which they have successfully interpreted the Editors wishes If there is some overlapping and if there are occasional differences of opinion these are not inappropriate in a symposium We thank also the publishers for their patience and within this Department we thank especially Miss Edna Hirst Miss Barbara Duckworth Mr Wilfred Lee and for the indexing we are indebted to Mr Reuben Silbermann We are particularly grateful to Mr Wade librarian of the Royal Society of Medicine for his translations and to those Editors from whose continental journals with their acquiescence the historically interesting descriptions of original operations have been extracted We hope our readers will find these classical contributions as interesting as we have done

Finally having written the above for both Mr Kyle and myself I must express my debt to him for his excellent editing without which progress would have been impossible and for his gracious Foreword I must thank Dr Lester Dragstedt the universally admired and beloved Chicago surgeon who has done so much to advance gastric surgery

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CHAPTER I

EPIDEMIOLOGY OF PEPTIC ULCERATION

By E. MAURICE BACKETT

PEPTIC ulceration is an increasing cause of morbidity. Indices by which we measure its prevalence are rising and this is not only because of the greater attention now given to the disease and the increased facilities which exist for its diagnosis.

Because mortality statistics for conditions like peptic ulcer reflect the quality of medical care as well as the amount and seriousness of the disease in the community they are suspect as indices of prevalence. However the fact that peptic ulcer is such an important cause of death (11 per 100 000 in 1957 in England and Wales) and that it is so much more important here than in many similar societies of comparable ethnic composition and diagnostic skill such as those of Western Europe (World Health Organisation 1955. Segi 1957) suggests that we in this country are either experiencing a higher prevalence or a more serious form of the disease. From a study in the United Kingdom by Doll, Avery Jones and Buckatzsch (1951) it has been estimated that between the ages of sixteen and sixty four years some 5 8 per cent of males and 1 9 per cent of females suffer at some time from peptic ulceration. This study showed that in 1951 the maximum prevalence in London was in middle aged males where nearly one in ten had an active or inactive peptic ulcer. In a survey of 3 223 consecutive necropsies which was undertaken by Dr Donald Teare a year later (Avery Jones 1957) the two ulcer types showed different epidemiological patterns with age. Duodenal ulcers were most common in middle aged men while gastric ulcers increased steadily in incidence with age. These findings are in agreement with several other surveys. Studies of prevalence in Norway (Knutsen and Selvaag 1947) and in Denmark (a variety of surveys discussed by Alsted 1953) suggest that there are no significant differences between these countries and England in the rate of diagnosed ulcer though it seems likely that Scotland has somewhat more. The United Kingdom however has by far the worst mortality experience of them all. Moreover in spite of improvements in diagnosis and treatment mortality shows no decline itself strong evidence of a continuing increase in the incidence or seriousness of the condition.

As well as its high cost in terms of death peptic ulceration causes a vast loss of working time and was responsible for over six million days of sickness among employed men in England and Wales in 1955. This was some three

per cent of time lost by men from all causes (Ministry of National Insurance 1955) The general practitioner must expect to spend up to four per cent of his working time in treating peptic ulcer (Backett Evans and Heady 1954) and several writers have drawn attention to its high and increasing cost in consultant time and diagnostic services

The increase

The epidemiologist has first to satisfy himself that the rapid increase in the reported occurrence of peptic ulcer is real and not the product of new medical techniques and interests Having satisfied himself that there has been a real and rapid increase he has sought to define the principal characteristics of those groups in our population which are most vulnerable

Various indices have been used which are likely to reflect the increase They include mortality rates (Morris and Titmuss 1944) and the reports of perforations and haematemesis (Avery Jones 1947 1955 1957) the perusal of ancient medical records for perforations (Jennings 1940) and an examination of discharges from the British Armies in two world wars (Morris *et al* 1944) Though of varying value all these analyses indicate a rapid increase Peptic ulcer which used to be a very rare disease at the end of the nineteenth century is now one from which every seventeenth man and fiftieth woman in our population is suffering or has suffered (Doll *et al* 1951) There seems no doubt that the most important increase during this century has been that experienced by middle aged men The most important decrease has been among young and middle aged women (Morris *et al* 1944 and the Registrar General 1936 also Jennings 1940 Illingworth Scott and Jamieson 1944 Avery Jones 1947 and others)

The increasing incidence of ulcer which is the most likely cause of the lack of decline in overall mortality (itself amazing in the light of surgical progress) is seen more clearly when age and site are considered Death rates for duodenal ulcer have remained steady and those for gastric ulcer have declined except among the elderly But because of the vastly improved care of all the complications of peptic ulcer these trends suggest an increasing incidence at both sites among the elderly and for duodenal ulcer among the young and middle aged The evidence indicates that there has been little change in the incidence of gastric ulcer in the younger group Avery Jones (1957) in summarising present trends is satisfied that improved diagnosis accounts for at least some of the apparent increase among the elderly but he is equally convinced of the reality of the increase in duodenal ulcer among the young and middle aged

With such strong evidence in favour of a real increase in ulceration it is necessary to consider the associated changes in the environment particularly of young and middle aged men It needs no epidemiological enquiry to see that in the last quarter of a century the way of life of a large section of the population has changed radically Changes in diet in occupation in leisure activities and in socio-economic status as well as in many other aspects of everyday life are some of the subjects that have been studied In addition familial regional climatic and ethnic differences in occurrence and severity have been analysed as have the possible causes of the differences between the sexes at various ages There follows a summary of what are considered to be the most fruitful of the recent epidemiological findings

The familial pattern

For many years it has been agreed that peptic ulcer occurs more frequently in the near relatives of ulcer patients than in the general population (Levin and Kuchuv 1936 Helweg Larsen 1946 Freeman 1947 and others) The details of this pattern were filled in by Doll and Buch (1950) and by Doll and Kellock (1951) who demonstrated a highly significant excess of ulcers in the siblings of patients with peptic ulcers and an only slightly less significant excess of ulcers in their fathers Mothers of ulcer patients however were found to experience no more peptic ulcers than the general population

In these two important papers not only is the familial nature of the condition demonstrated but the all important question—from a genetic point of view—of site specificity is answered It was shown that there is an excess of gastric ulcer among relatives of patients with gastric ulcer and of duodenal ulcer among relatives of those with duodenal ulcer This concordance is significant between siblings and from generation to generation and is independent of sex or social class The family pattern of ulceration described by Doll and Buch and other workers follows no simple genetic hypothesis though it does suggest independent mechanisms in the inheritance of the two ulcer types The genetic component or inherited tendency is likely to follow a complicated genetic pattern It does not of course exclude environmental factors for example it has been noted that the relatives of peptic ulcer patients who were the subjects of these studies shared more than a blood relationship with the affected members A similar home environment was also shared though it is difficult to see how this common background could affect the site specificity The genetic factors which are operating would seem to do little more than predispose certain individuals to one or other or both types of ulcer The mechanism of predisposition has been investigated by work on the association of ulcer and certain blood groups and secretor

mechanisms For many years it has been known that both duodenal and gastric ulcer are significantly more common among persons with blood group O In considering the results of many surveys Roberts (1957) shows that roughly the same association between gastric ulcer and group O exists for different areas while duodenal ulcer shows some interesting geographical variation being small in for example Glasgow and Vienna The association of blood group O with duodenal ulcer is stronger than with gastric ulcer and is the same for both sexes at all ages A further advance (Clarke *et al* 1956) was the demonstration of the association of duodenal ulcer and the inability to secrete substances with ABO specificity in body fluids These studies suggest that a specific protection from duodenal ulceration perhaps at a cytological level might be associated with secretor ability in males and females Non secretors appear to be some forty five per cent more likely to develop ulcers than secretors and group O non secretors are more than twice as liable to develop duodenal ulcers than are secretors of groups A B and AB (Clarke *et al* 1959) The factors which determine whether the genetically predisposed individual ultimately becomes an ulcer patient are not yet known Available evidence suggests that the final steps depend upon the interaction of a group of environmental forces which are at the moment responsible for the rapid changes in incidence and in social class occupational and geographic patterns

The sex ratio

About twice the number of gastric ulcers and between four and seven times as many duodenal ulcers are found in men as are found in women Sex ratios based upon hospital figures from which so much of the information about peptic ulcer comes will not represent this true state of affairs but will be distorted by severity and the social and economic factors associated with referral or admission to hospital The sex ratio at perforation or death both of them sources of information in which much confidence may be placed will of course reflect the relative seriousness rather than the true prevalence of ulceration in the population Most early studies which estimated prevalence suffered one or more of these distortions and the assumption that for both gastric and duodenal ulcer a maximum sex ratio occurred in young middle age is probably wrong It now seems likely (Avery Jones and Doll 1953) that there is a constant ratio at all ages for each site

Both gastric and duodenal ulcer are more serious threats to men as well as being found more frequently among them Thus a peptic ulcer is nearly twice as often fatal in the male as in the female (Doll 1958) Regional differences in the sex ratio—for example the high proportion of both gastric and duodenal ulcers among women in Scotland—are not yet explained

Two features of the occurrence of peptic ulcer in women emphasise the already well known importance of endocrine factors in aetiology There is a sharp increase in peptic ulcer after the menopause and there is a striking deficiency of peptic ulcers (other than those associated with hiatus hernia) during pregnancy (Sandweiss *et al* 1943 and Avery Jones 1947)

Town and country

In spite of medical care being less readily available in rural districts at least as far as surgical emergencies are concerned the death rate from peptic ulceration is uniformly lower than in towns at the age of thirty five years and above Small towns show a better experience than large towns and London has the worst experience of all' (Registrar General 1958) Differences in prevalence have not been demonstrated but Doll *et al* (1951) and others have found a deficiency of peptic ulcer among agricultural workers and it seems likely that the death rates reveal a true difference between town and country The difference could result from a drifting of ulcer patients from the country to the relatively lighter jobs in the towns However this drift though it may occur is unlikely to be enough to account for the differences which are described

Marital state

The important excess of deaths from peptic ulcer (and some other diseases) at all ages among the divorced and the somewhat less striking excess among widowed and single persons when compared with married persons was demon strated recently by the U S Department of Health Education and Welfare (1956) These differences in American mortality experience were found only for men For women marital status did not appear as significant There is no con firmation of a comparable pattern in Great Britain but such variations if con firmed would constitute an epidemiological clue from which could stem the studies of such variables as eating and smoking habits as well as other differ ences between the groups concerned Already these variations have been used without adequate control in support of certain psychological hypotheses in aetiology

Social class

Classifications of men according to the Registrar General s five social classes result in the grouping together of those of comparable prosperity and job status Thus the professions (Social Class I) are distinguished from the skilled occupations (III) which are in turn distinguished from the unskilled

occupations (V) Social classes II and IV occupy intermediate positions Such a classification shows a steady gradient in the *prevalence* of gastric ulcer with an increase from Social Class I to Social Class V and no significant gradient for duodenal ulcer (Doll *et al* 1951)

In this respect as in several other important characteristics the two diseases appear epidemiologically distinct and this distinction is also reflected in the mortality experience in the various social classes (Registrar General 1958) Deaths from peptic ulcer follow a somewhat similar trend those due to gastric ulcer rise sharply from Social Class I to V and those certified to duodenal ulcer show a less certain gradient When these trends are considered in conjunction with the changing incidence of the last half century two notions may be derived First duodenal ulcer appears to be responding to changes operating more equally throughout all classes of society and second gastric ulcer with its impressive social gradient may be more intimately linked with poverty or with some aspect of the poverty complex of behaviour attitude and way of life It has been suggested that the social gradient shown by deaths from gastric ulcer is a product of differences in the quality of medical care between the different social groups Another explanation has been that there are sufficient genetic differences between classes to ensure a social class pattern for reasons of heredity Neither explanation is adequate by itself first because of the social gradient in the *prevalence* of gastric ulcer mentioned above and second because blood group and secretor differences between social classes are not striking

Occupation

The classification by occupation of patients with peptic ulcer has been attempted in several European countries as well as in the United States and South America The most comprehensive study in Great Britain is once again that of Doll *et al* (1951)

In this study occupations were classified into twenty large groups and the expected number of ulcer patients to be found in each was calculated from the experience of all those taking part in the survey Occupations with a significantly higher prevalence than was expected are by now familiar to all They were the so-called industrial tension jobs of foremen business executives and others who hold positions of responsibility in industry Doctors also showed the well known excess and notable deficiencies were among unskilled sedentary and agricultural workers Among those occupations whose experience of ulcer was near to that expected for the whole population were transport drivers and conductors a group formerly thought to be especially vulnerable In this as in other matters gastric and duodenal ulcer showed

different patterns The excess found in the industrial tension jobs and among doctors was due almost entirely to proved duodenal ulcer In commenting on these and other studies Doll *et al* (1951) and Avery Jones (1957) consider the excess among doctors to be due to better diagnosis rather than to a greater prevalence among the medical profession It is important to recognise that persons in positions of industrial responsibility may share more than the tensions of their job Other features of their lives in common to them all may be found to play a part in aetiology

The timing of perforations

Because of the acute and memorable nature of a perforation accurate records have been kept of the months of the year the days of the week and perhaps somewhat less accurately the hours of the day of their occurrence In addition some attempts have been made to relate the timing of perforation to external events and particularly to the occurrence of air raids during the late war

A study in Scotland by Illingworth Scott and Jamieson (1944) drew attention to certain peak periods for perforation Average monthly incidence charts pointed to the winter as the worst period of the year with the late summer as the best There was a marked and significant decline at the end of each weekend (Sunday and Monday showed the smallest number of perfora tions) and the daily peak was in late afternoon with the best period in the morning This study also drew attention to the increase in perforations in 1941 *prior* to the bombing of Glasgow and the significant drop in 1943 The authors favour a causal relationship between the perforations and the un doubted disruption of normal routine the excess of overtime the lack of sleep and the irregular meals which were widespread at the time

A study of the effect of the London blitz (Stewart and Winser 1942) showed a striking association in time between air raids and a sharp increase in the monthly rate of perforations Similar events (though not so well docu mented) were reported from Bristol Liverpool and Newcastle

Associations in time are notorious for their ability to delude but the dislocation of normal habits and above all the fatigue of the early war years find support as aetiological factors in the monthly weekly and daily patterns Common themes are activity and rest Doll *et al* (1951) found irregular meals and shift work unimportant in the aetiology of ulcer itself Certainly further study of the mechanism of perforation might assist our understanding of the ulcer process

Ulcer and smoking

In their well known studies of lung cancer and other causes of death among doctors in relation to their smoking habits Doll and Hill (1956) showed

a steady increase from non smokers to heavy smokers in deaths from peptic ulcer tuberculosis chronic bronchitis coronary disease and lung cancer A similar dose response relationship has been shown by Brown McKeown and Whitfield (1957) and others

The association between peptic ulcer and chronic pulmonary disease has also been demonstrated in life (Weber and Gregg 1955 and others) The association in death has been shown by many workers for example Avery Jones (1957) on Dr Teare s necropsy material In this series there was a significant excess of both gastric and duodenal ulcers among persons whose death was associated with bronchitis the largest excess being in the duodenal ulcer group

It is not possible to be sure whether these associations are because of smoking It is possible that secondary relationships are involved but the suggestion of a direct or primary relationship with smoking is very strong The excess of peptic ulceration in Scotland may be related to the consumption of tobacco in that country

Psychological factors

Epidemiological study of the operation of psychological factors in the aetiology of peptic ulcer has been dogged by three major difficulties First the unrestrained enthusiasm of the advocates of psychological causation second the difficulty of defining the psychological variable with the necessary clarity and objectivity and third the difficulty in retrospective studies of measuring the effect of the ulcer itself upon the personality under investigation The two latter difficulties are being overcome to some extent by the use of combined objective psychiatric and case work methods (Goldberg 1958) Statistically there is a strong and significant association between ' stress and anxiety of different sorts and peptic ulcer—mostly duodenal (Doll *et al* 1951 Gainsborough Slater 1946 and others)

It has been suggested that the idea of a psychological component in the aetiology of peptic ulcer receives support from the differences in the prevalence of duodenal ulcer between urban and rural areas and from its occupational pattern The first because it is supposed—without evidence—that a rural life involves less stress and second because of the proved association of ulcer and responsible jobs The high incidence of ulcer in the divorced widowed and unmarried male is also mentioned and it is suggested that the married state is less stressful than any of these Emotionally charged events in the lives of ulcer patients have long been associated by clinicians with perforation and haemorrhage (Davies and Wilson 1939 and Avery Jones 1947) but more evidence is needed before this association is established Certainly the grosser forms of disturbance in early life have no effect (Kellock 1951)

Evidence of association of ulcer and anxiety and of perforation and bombing supports the hypothesis that emotions play a large part in the lives of ulcer patients Such a hypothesis is certainly helped by clinical impression and popular comment but support of a less transient nature is needed as well as the demonstration that the disturbances and patterns of behaviour so often described as typical do not derive from peptic ulceration itself In short while the treatment of the ulcer patient demands insight into his characteristic problems and disturbed personality—and he may share with other patients similar patterns of upbringing—there is as yet no more than circumstantial evidence that these are contributory causes of his ulcer

Summary

In this brief review of the epidemiology of peptic ulcer attention has been drawn to the proved and striking increase of the condition in recent years an increase pointing to the operation of environmental factors in its aetiology Peptic ulcer though no more common than elsewhere in North West Europe is a more serious disease in Great Britain By middle age one in seventeen men and one in fifty women have or have had a peptic ulcer but women are protected by pregnancy and suffer much more peptic ulceration after the menopause than before

A site specific predisposition to peptic ulcer is inherited and there is an association with the O blood group and with an inability to secrete ABO substances in the body fluids Predisposition to duodenal ulcer appears to be stronger than to gastric ulcer

Clues as to the environmental factors in aetiology come from studies of occupation social class urban and rural life from the timing of some of the complications of peptic ulcer and its distribution among people of different psychological types and marital status Cigarette smoking appears to be an important factor in predisposed individuals

The vulnerable individual is likely to have one or more of the following characteristics he will have near relatives who have a similar type of ulcer he will live in a large town rather than the country and he will be employed in industry in one of the so-called tension jobs *i e* he will be a foreman an executive or at least a person with some responsibility If he is from one of the lower socio-economic classes he is more likely to have a gastric ulcer than if he comes from a higher social class duodenal ulcer however is almost equally distributed across the classes Finally he may be a bronchitic and a heavy smoker and he may show unusual emotional and personality reactions and anxiety Should his ulcer perforate or bleed he may suffer these complications at a time of disturbance or stress and he will be most

likely to perforate in the winter and during the week rather than at the weekend and towards the end of the day rather than after rest

There is growing evidence based upon information as to the familial pattern of peptic ulcer as well as upon almost all recent epidemiological studies that gastric and duodenal ulcer are in their mass aspects at least separate and distinct diseases

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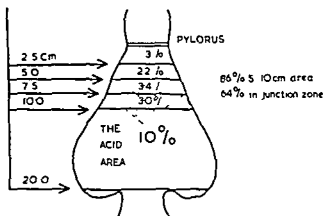
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CHAPTER II

THE AETIOLOGY OF PEPTIC ULCER

By WILFRED SIRCUS

THE essential mechanism in the formation of a peptic ulcer is the digestion of the alimentary mucosa by acid and pepsin There are many factors which may activate this mechanism and in any individual with a peptic ulcer more than one of them may be involved The purpose of this chapter is to examine the various factors which may play a part in the production of an ulcer The information gained from experimental studies on animals is considered first The problem of chronicity is then examined and the reasons



THE SITING OF GASTRIC ULCERS

FIG 1

for the natural resistance of the mucosa to peptic digestion and the role of the duodenum and of the alkaline juices in the control of acidity are discussed The relation of the nervous system and of stress in particular to the function of the stomach and duodenum is also considered Perhaps most important of all the evidence is that obtained from a study of the abnormal alimentary physiology in humans with peptic ulceration and the final section examines newly acquired data

The majority of peptic ulcers which occur in man develop in the mucosa of the alimentary tract outside but close to that part of the stomach which contains the parietal cells Ulcers within the parietal cell area constitute only fourteen per cent of the total incidence of gastric ulcers (Fig 1) Thus almost

wards The incidence of ulceration in the jejunum parallels the resulting change in pH but the ulcer develops at the point on the intestinal mucosa where the jet of gastric juice first impinges It can be shown that the rate of development of the ulcer is related to the propulsive force of the jet of juice The lesion in the Mann Williamson animal is identical with that complicating gastrojejunostomy in man

By progressively shifting a gastro intestinal anastomosis down the small intestine (Merendino *et al*, 1945) it has been shown that the incidence and rapidity of ulceration is greater the more caudal the anastomosis is placed As the anastomosis is moved down the ulcer in the jejunum at the site of the previous stoma heals and a further one forms opposite the new anastomosis The severity and incidence of experimental anastomotic ulcer is directly related to the load of acid gastric juice the intestine has to resist Progressive resection of the area bearing the parietal cells results in decreasing incidence and severity of anastomotic ulceration (Sircus (1) 1956) Conversely exposing the duodenum to the full effects of unbuffered gastric juice by the formation of an innervated pouch of the whole stomach with oesophago jejunal anastomosis (Winkelstein *et al* 1956) results in the rapid death of the animal from perforating duodenal ulcer

Hypersecretion ulcer

Chronic peptic ulcers identical with those resulting from anastomotic procedures can readily be produced without anatomical interference if prolonged stimulation of acid gastric juice is attained by the administration of histamine carbaminoyl choline reserpine priscoline caffeine *ACTH* or cortisone This has been shown in man (McHardy and Brown 1944 Horton *et al* 1944) and in several laboratory and more exotic species (Code and Varco 1940) It has been shown in the dog that if *the stomach secretes* at or close to its maximum capacity for five to ten days chronic ulceration results The continuous flow into the stomach of 1,800 to 2 400 ml of 0 15N HCl is necessary *(Fogelman et al 1949) and it is of interest* that this large quantity of acid does not slough away the pyloric or duodenal mucosa but results in the formation of circumscribed duodenal ulcers thus suggesting *that some factor of localised impairment of defence is involved*

Toxic ulcer

Cincophen poisoning *in man has been complicated* by peptic ulceration (Bloch and Rosenberg 1934) and peptic ulcers have appeared after or been aggravated by the administration of salicylates butazolidine and caffeine *These substances* together with pitressin and pilocarpine will frequently

produce ulceration in the stomach of laboratory animals if administered in near toxic dosage With cincophen there is a general systemic upset with an initial diffuse haemorrhagic gastritis followed by multiple subacute mucosal ulcers one or more of which gradually assumes the typical appearances of a chronic peptic ulcer It has not been shown that stimulation of acid secretion is the mechanism of production of ulcers in these circumstances That the presence of acid is essential is suggested by the value of the simultaneous administration of antacids in preventing ulcers in the main stomach and by the development in the same animal of ulcers in fundic pouches the contents of which are unbuffered by food or alkalies (Stalker *et al* 1936 1937) Multiple haemorrhagic erosions leading to ulceration also appear to be the characteristic effects of the other agents mentioned Caffeine induced ulceration however is associated both with definite stimulation of acid secretion and local vascular congestion (Roth *et al* 1944)

HISTOPATHOGENESIS OF CHRONIC PEPTIC ULCERATION —FORCES OF ATTACK AND DEFENCE

Chronic peptic ulceration appears to develop as a result of the continuous operation of certain factors upon an antecedent acute ulcer so that healing fails to occur This belief is supported by several observations The anastomosis of a gastric pouch to a segment of the jejunum opening on to the abdominal wall permits naked eye observation through a sigmoidoscope of the various stages in the development of a chronic ulcer (Harper 1935) The same progressive changes are seen in the duodenum after continuous histamine administration in the dog and in man (Horton *et al* 1944) Taylor and Warren (1956) have shown that perforated acute peptic ulcers in man heal without sequelae if treated conservatively but if sutured frequently develop into indolent chronic ulcers at the same site the sutures presumably acting either as irritants or interfering with the local blood supply In cincophen toxaemia the progress from haemorrhagic gastritis to acute erosions and subsequent chronic peptic ulcer is clearly seen Significantly although the initial subacute cincophen lesions are widespread in the stomach the final chronic ulcers are invariably located outside the parietal cell area in the pyloric part of the stomach (Bollman *et al* 1938)

The presence of a simple break in the mucosa does not *per se* result in peptic ulceration Peptic ulcers have not been observed to develop at the site of gastric or duodenal suction biopsies and within five days of the appearance of gastric erosions causing severe haemorrhage the mucosa may be quite normal on inspection through a gastroscope The rate at which gastric epithelium regenerates may be extremely rapid under normal conditions In

the cat it has been shown that within a few hours complete regeneration may occur of mucosa cut back to the level of the necks of the glands (Grant 1945) Nevertheless a break in the mucosa as the result either of traumatic disruption or of devitalisation is a necessary precedent to a chronic ulcer The only ulcers in the formation of which traumatic disruption of the mucosal surface is likely to be of importance are those seen in the vagotomised rabbit fed roughage and those present in numbers in the stomach of the Californian seal This animal habitually swallows sharp larval stones to facilitate trituration (Schroeder and Wegeforth 1935)

In the absence of trauma something must cause preliminary devitalisation of the mucosa to institute the acute ulceration and the process must operate for a sufficiently long time to permit the ulcer to become chronic At this point it is useful to assume that the integrity of the lining of the digestive tract depends upon the balance of two forces the tendency of the acid and digestive enzymes to bring about autolysis and the properties of the mucosa and of mucus which protect it from this Autodigestion is seen in life as when the pancreas is injured or its blood supply reduced, or in the mucosa of a loop of gut with a strangulated venous outflow and in the alimentary tissues after death It has been observed in the mouse that the intestinal cells are digested in the fasting state and regenerate as soon as feeding recommences (Sun 1927) The resistance to autolysis is a special property of adaptation possessed by tissues normally exposed to contact with enzymes Thus while the skin around a gastric or intestinal fistula is rapidly digested the external covering of the ascaris or the tapeworm is unaffected by enzymes so enabling them to survive in the gastro intestinal tract

The attacking force in chronic peptic ulceration is the acid pepsin containing gastric juice Acid devitalises tissue and peptic activity removes the devitalised cells thus exposing deeper tissue to the further action of the acid (Matthes 1893) Thus it is possible that the exposure of mucosa with normal defensive properties to unusually severe attack by acid pepsin or the exposure of mucosa with inadequacy of defensive properties to a normal force of attack may equally result in the destruction of mucosa which is the necessary precedent for chronic ulceration

The Forces of Defence

The protective factors in mucosal resistance

The resistance of certain cells to digestion by enzymes seems to depend upon the chemical composition of the cell surfaces The surface membrane of the epithelium of the gastro intestinal parasite worms is deficient in protein

and peculiarly rich in lipoids Hydrochloric acid penetrates only slowly into cells Strong acids will cause the death of muscle cells when the pH drops below 5 yet alkalies do so only when the pH rises above 11 As the pH of the intestinal tract rarely reaches 9 0 it becomes clear why peptic but not tryptic ulcers occur On a cell equivalent to a 10 μ cube one surface layer will contain 10 000 molecules of protein and the entire cell one million (Northrop 1926) Thus surface digestion would account for only one hundredth of the cell content and it is reasonable to suppose that the cell could effect continuous repair However according to Hollander (1954) the mucosal barrier to digestion has two components the layer of viscous mucus and the columnar cells lying below the mucus and lining the crypts of the glands He has demonstrated that the destruction by eugenol of the surface layers of the mucosa of the stomach is made good in thirty six hours by the reformation of columnar cells and crypts The defensive value of mucus is attributed to five properties the tendency to adhere to the underlying cells the cohesiveness which allows it to form an uninterrupted sheet over the stomach the viscosity which resists flow and the absorptive power for pepsin and buffering power for acid It is of interest in relation to this that after six days of administration of A C T H gel to volunteers Hirschowitz (1956) found a marked decline in the output of mucus and in the viscosity of the gastric juice In two of the volunteers ulcers developed in the stomach four days after commencing the hormone Alteration of the output of acid and pepsin had not been observed up to the time of development of the ulcers The healing of these ulcers coincided with a sharp rise in the output and viscosity of the mucus and a disappearance of acid and pepsin from the juice Further evidence of the importance of mucus in the resistance to digestion is provided by Griffiths and Harkins (1956) who dropped a mixture of hydrochloric acid and pepsin on to the exposed mucosa of the duodenum and jejunum of dogs and pigs after diverting the bile and pancreatic outflows After a standard exposure there was considerable desquamation and necrosis of most of the duodenum and of the jejunum of the dogs and of the jejunum of the pigs The whole of the duodenum in the pig however was only slightly affected as was the first two centimetres of the duodenum in the dog The morphological differences between these areas of the two species lies in the distribution of the glands of Brunner In the dog they extend distal from the pylorus for only two centimetres whereas in the pig they form a dense sheet down to the biliary ampulla as is the case in man Segments of pig s duodenum implanted between the stomach and loop of jejunum have likewise been shown to resist ulceration whereas segments of ileum do not (Florey *et al* 1939) Hence it appears probable that either of the following factors will lower the defences against the attack of acid pepsin (*i*) a deficiency in

the production of mucus in the stomach or duodenum by reason either of a reduction in the number or secretory efficiency of the columnar cells or Brunner's gland cells or (*ii*) an alteration in the chemical character of the surface membrane of the columnar cells

Vascular factors

Anoxic alimentary tissue loses its capacity to withstand digestion by enzymes Hence reduction of the flow of the blood through mucosa exposed to acid pepsin would be conducive to peptic ulceration There is no satisfactory evidence concerning the mechanism of the initial devitalisation of the cells beneath the mucus which permits peptic removal The belief that ischaemic necrosis from vascular damage is responsible has often been stated based largely on the observed effects of pilocarpine (Westphal 1914) histamine (Williams 1951) and pitressin (Crane 1954) in producing multiple areas of focal necrosis in the stomachs of small laboratory animals While damaged capillaries in the affected foci were observed by Watt (1956) in the histamine stimulated mucosa of the guinea pig stomach he found that these changes could be prevented by the simultaneous perfusion of the stomach with weak alkali or saline He suggests that the effects observed in the vessels are secondary to the permeation of the mucosa by acid the real problem is to explain why this focal penetration by acid occurs

The absence of end arteries and the presence of very rich vascular anastomoses in the mucosa of the human stomach render it unlikely that simple embolic or thrombotic factors could be responsible for devitalisation However the demonstration by Barclay and Bentley (1948) of arterio venous shunts in the submucosa makes it possible that neuro vascular phenomena may operate in certain conditions to produce areas of ischaemia in the gastric mucosa Lesions in the brain stem and hypothalamus and intracranial operations may provide such conditions and are sometimes associated with erosions in the stomach and duodenum

Nutritional factors

Reported geographical and racial differences in the incidence of peptic ulcer have led many to suppose that dietary deficiencies and especially states of subnutrition lead to a lowering of the alimentary defences against peptic ulceration (Kouwenaar 1930 Eagle and Gillman 1938 Somervell 1942) but for lack of adequate data or controls none of the reports survives scrutiny and all await confirmation Experiments designed to reproduce the deficiencies of nutrition have consistently failed to produce peptic ulceration (Orr and Rao 1939 Dogra 1941)

In the Registrar General's returns mortality statistics for peptic ulcer do show an inverse relationship to social status but too many factors other than nutrition are involved in this association for any conclusions to be drawn There was no general increase in the incidence of peptic ulcer in Europe during the war years despite the privations suffered by many nations

The role of hormones

One of the most remarkable but ill understood examples of protection against peptic ulceration is that of the beneficial effect of pregnancy The protection against digestion of a colonic implant into the stomach of a pregnant bitch demonstrated that the changes providing the increased power of defence are not confined to the gastric or duodenal mucosa (Sircus (i) 1956) Neither is it a factor of altered acidity of the gastric juice for it has been shown that in dogs and in human subjects acid output is undisturbed in pregnancy and is increased in lactation (Clarke and Tankel 1954 McCarthy *et al* 1954) It has been claimed that the urine of pregnant women contains much greater quantities of an ulcer preventing substance than that of the non pregnant women or of men and that there is less than normal in the urine of subjects with peptic ulcer (Sandweiss *et al* 1952) This substance named anthelone is said to be present in extracts of urine which protect Mann Williamson dogs against peptic ulceration without concurrently affecting gastric secretion (Hands *et al* 1942) When injection of urine extracts causes depression of gastric secretion and to some extent motility another substance urogastrone is held to be responsible (Gray *et al* 1942)

Both anthelone and urogastrone have been thought to be the urinary excretion products of enterogastrone a material recovered from homogenised mucosa of small intestin and which is capable of inhibiting gastric secretion and motility (Kosaka and Lim 1930 Gray *et al* 1937) It is tempting to suppose that in certain individuals there is a deficiency of a protective hormone (which the above substances represent in some fashion) which makes these individuals prone to peptic ulceration and that in pregnancy there is an increased activity of this protective factor However although the evidence is sound enough for the existence of extracts of gut and urine which depress gastric secretion there is no real confirmation that they will protect humans or experimental animals against peptic ulceration There is confusion over the identities of enterogastrone urogastrone and anthelone and no single substance has been isolated so that its chemical nature may be determined But more exact methods of extraction of urine are now available and are productive of highly active material capable in small concentration of inhibiting gastric secretion (Gregory 1956) A new lease may be given to this sector of inquiry into the problem of peptic ulcer and may lead to the elucidation

of the defensive side of the 'equation of ulcer aetiology' (acid + pepsin) v (mucosa resistance) (Card 1952)

The action upon gastric secretion of A C T H and cortisone like steroid hormones and the relation of these substances to peptic ulceration remains ill understood Conflicting reports are published of the effects of administration of the drugs Some show that gastric secretion and the tendency to peptic ulceration are increased, while others appear to demonstrate a depression of

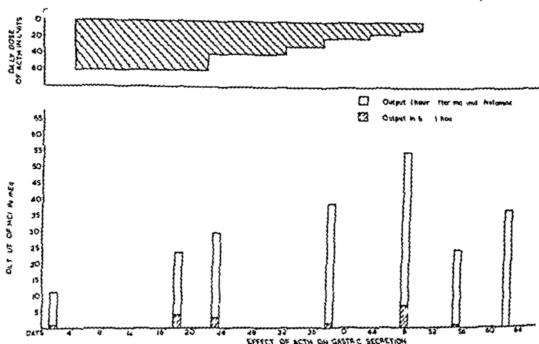


FIG 3

all the elements of the gastric juice In the Gastro-Intestinal Unit at Edinburgh clear evidence has been obtained of remarkable increases in the maximal response to histamine after prolonged administration of the hormones in the treatment of colitis (Card Crean and Sircus unpublished data) (Fig 3) But it has not been conclusively shown that the incidence of peptic ulcer in subjects being treated with the hormones is greater than that of an untreated comparable population and where ulcers have appeared to complicate steroid therapy a high proportion have been gastric in site This suggests an absence of relation between any ulcerogenic and acid stimulating properties (Wollaeger 1954 Kammerer 1957) Only one case of fresh peptic ulcer has appeared in a substantial number of subjects at Edinburgh treated for ulcerative colitis with high doses of steroid hormones continued in some cases for several months and despite measured increase in the gastric response to histamine In the one developing ulceration there had not been any alteration of gastric secretion while on the hormone

The mechanism involved in the potentiation of gastric secretion by steroid hormones is unknown However it has been shown that the activity of the enzyme d amino-oxidase which takes part in intracellular oxidation reduction reactions is increased by cortisone and depressed by desoxycorticosterone (Umbreit 1951 Hayans and Dorfman 1951) Kyle (1956) noting this and that cortisone increased gastric acidity while desoxycorticosterone depressed it suggested that the site of action of the hormones in the stomach was the enzyme systems within the parietal cells He thought that the function of the hormones was to maintain the cells of the gastric mucosa in an optimal state for reacting to stimuli Support for his belief is provided by our observa tions that in man the prolonged administration of cortisone or A C T H rarely has any influence on the basal resting secretion of the stomach but commonly upon the response to stimuli such as the injection of histamine (Crean unpublished data)

The role of the duodenum and the alkaline juices

CONTROL OF GASTRIC SECRETION BY THE DUODENUM —It has been shown using dog preparations that a homeostatic mechanism operates both from the antrum (Harrison *et al* 1956) and from the duodenum by which a critical level of concentration of H ions in the alimentary contents causes the inhibition of further secretion of acid by the stomach In the duodenum of the dog both a neural and a hormonal mechanism operate the former activated by acid and the latter by hyperosmolar solutions and by products of the digestion of fats (Sircus 1958) Excision of the duodenum in the dog or transplantation to lower down the alimentary tract results in a great increase in the gastric secretory response to meals (Brackney *et al* 1955) It is conceivable therefore that interference with these mechanisms by local structural or metabolic disorders might result in the uninhibited production of acid both in response to meals and in the fasting state which could lead to peptic ulceration

A few studies on subjects with duodenal ulcer have demonstrated some disturbance in the acid activated duodenal mechanism for inhibiting gastric secretion (Shay *et al* 1942 Sircus (2) 1956 Hunt (1) 1957) But the results are not highly significant and they are not found in a sufficient proportion of the ulcer subjects to ascribe aetiological importance to the phenomenon Abnormalities in gastric emptying or abnormalities in the purely osmotic regu lation of gastric secretion do not appear to play any part in the pathogenesis of ulceration (Booth *et al* 1957 Hunt 1957 (2)) When the antrum is separ ated from the rest of the stomach by a mucosal barrier and the body drained by a gastro-enterostomy the secretory response of the stomach in the fasting

state to meals and to hypoglycaemia rises markedly (Uvnas *et al* 1956) Some of this effect may be the result of failure to activate the pH *sensitive* antral mechanism for controlling acid production and some to increased production of gastrin from the antrum in the presence there of a prevailing alkaline pH A further possibility is the facilitation of subthreshold vagal and other stimuli which may be involved in active gastric secretion It is noteworthy in this connection that the injection of histamine in beeswax into dogs after resection of the antrum and half the body of the stomach was followed in most cases by the formation of duodenal ulcers whereas retention of the antrum prevented this result in most dogs (State *et al* 1955) Hence some disturbance of the antrum may likewise play a part in the abnormal physiology conducive to or associated with peptic ulceration This theory remains unproved

THE BILIARY AND PANCREATIC SECRETIONS —Experimental studies on dogs with obstructed bile ducts and pancreatectomy have shown that any increased tendency to formation of duodenal ulcers on prolonged stimulation of acid is associated with the interference in nutrition resulting from the procedures and is not related to changes in the levels of intraduodenal acidity or to acid base imbalance in the blood (Mann and Bollman 1932, Berg 1934)

In cases of duodenal ulcer in man there is no evidence of any deficiency in the production of alkaline neutralising juices Furthermore it has been shown that the general pattern of intraduodenal acidity throughout the twenty four hours closely reflects that in the stomach and is largely independent of the output of biliary and pancreatic secretions (Atkinson and Henley 1955)

The role of the nervous system and stress

Since the classical observations on the effects of emotion upon the exposed gastric mucosa of Alexis St Martin (Beaumont 1833) numerous studies have appeared on the relation of emotion to gastric function but few have any scientific value One most useful study (Kellock 1951) made a comparison of childhood social and emotional factors in 250 cases of duodenal ulcer and an equal number of subjects with other diseases Factors investigated included the size of the family the social class the presence or otherwise of domestic stress the history of illness and of educational attainment Kellock found no difference whatsoever between the two groups and no evidence to support the concept of an ulcer personality

Two major hypotheses have been proposed to explain a relationship between the disturbed mind and peptic ulcer One states that repressed and frustrated dependency feelings give rise to an emotional disturbance with

a desire to be fed and through that to chronic activation of the para sympathetic nervous system (Alexander 1950) The other states that any emotional conflict without specificity gives rise to chronic anxiety which in turn produces chronic stimulation of the vagus nerve (Mahl 1950) The evidence from the outstanding study on the subject Tom (Wolf and Wolff 1943) fits both concepts in that emotional reactions involving conflict morbidity resentment and anxiety were accompanied by increased secretion motility and blood flow in the stomach During such situations local trauma or repeated removal of surface mucus while applying hydrochloric acid resulted in the formation on the gastric mucosa of erosions and small punched out ulcers In laboratory animals experiments designed to produce chronic conflicts have not led to the development of chronic peptic ulcers although haemorrhages and superficial erosions can readily be observed During the war years 1940-43 the incidence of perforations of peptic ulcers rose sharply in the U K and in Scandinavian countries (Stewart and Winser 1942) but it occurred in uncommitted Sweden and could not be correlated with the stress of aerial bombardment as originally suggested However there is good reason to suppose that general public stress was as great in Sweden whose neutrality was continually threatened as in the other countries where the phenomenon was observed From these experiments and experiences it may be accepted that psychological stress can result in acute changes in the gastric and intestinal mucosa but no evidence has appeared to prove a relationship to chronic ulceration

The same difference emerges from a consideration of the relationship between peptic ulcer and damage and disorder of the brain In over 1 000 cases of brain lesions known to Cushing (1932) only one was complicated by a really chronic peptic ulcer and in 500 cases of pituitary tumour only five had an associated peptic ulcer (Wilson *et al* 1946) The alimentary changes associated with brain stem lesions and with hypothalamic trauma are acute ulcers invariably beginning as focal haemorrhages An important factor in the genesis of such complications appears to be the degree of associated shock The prolonged stimulation of the vagi or the administration of para sympathomimetic drugs to laboratory animals have rarely resulted in chronic peptic ulcer even where the vagi have been electrically stimulated many hours each day for several months Thus it is acceptable that erosive and ulcerative gastro intestinal disorders may be a by product of the process of adaptation to stress but that these are usually acute and superficial in nature In the mediation of such influences two pathways could be involved The first is that from the hypothalamus directly down *via* the vagus nucleus to the vagus nerve and the viscera The other is that from the hypothalamus to the anterior pituitary resulting in the secretion of A C T H and thus the stimulation of

a secretion of corticoids from the adrenal cortex Stimulation of the anterior hypothalamus in monkeys initiates gastric secretion which reaches its maximum in half to one hour and is blocked by prior vagotomy Stimulation of the posterior hypothalamus initiates gastric secretion with a maximum effect at two and a half to three hours and is prevented by prior adrenalectomy Insulin induced hypoglycaemia in these animals has effects on gastric secretion with characters of both the above mechanisms and an appropriate part response can be obtained by carrying out beforehand either vagotomy or adrenalectomy Furthermore whereas the injection of A C T H and cortisone produces similar secretory changes to those observed after stimulation of the posterior hypothalamus the A C T H effect is present after vagotomy but absent after adrenalectomy (Porter *et al* 1953)

Considerable overlap must exist between the different mechanisms operating to stimulate gastric secretion Dragstedt (1956) has noted the great rise in acid output occurring in subjects in the twenty four hours before an expected operation and in the Edinburgh Gastro intestinal Unit the same phenomenon has been observed to influence the maximal histamine response The synergism of the hormonal and the neural mechanisms of gastric secretion is well established (Uvnas 1942) Dragstedt s claim that the characteristic high acid output in cases of duodenal ulcer is due solely to high vagal tone and is completely abolished by vagotomy is not substantiated by the experience of others Analysis of the data of Dragstedt *et al* (1950) shows that the percentage diminution of nocturnal secretion by vagotomy was the same in cases of gastric ulcer as in those with duodenal ulcer so that the vagal tone appears equally important in maintaining basal output in both groups (Marks 1959) The response to maximal histamine stimulation is considerably reduced by vagotomy but in a substantial number of cases with recurrent ulceration after gastric operation the basal fasting secretion remains much above normal despite vagotomy and even where prior gastrectomy has removed the antrum and much of the parietal cell mass as shown by the response to histamine (Card Crean and Sircus unpublished data) The high basal output in cases of chronic duodenal ulcer is probably the resultant of several factors of which vagal tone is only one One possible mechanism for the prolonged stimulation of acid through either neural or neuro hormonal pathways is suggested by the results of present studies in both humans (Crean Huston and Sircus 1959) and dogs (Huston and Sircus 1959) In both species acid secretion of remarkable quantity and duration has been shown to occur subsequent to the production of hypoglycaemia with insulin but several hours after the blood sugar has returned to a normal level and the initial phase of gastric secretory response has ceased

In subjects with duodenal ulcer estimations of plasma corticoids and 17 ketosteroids which might have been expected to demonstrate higher than normal levels if chronic stress was of importance in aetiology did not in fact show any difference from those in subjects with other diseases (Freeman *et al* 1956) It may be that examination of the urinary excretion of these hormones in subjects with peptic ulcer may be more revealing as methods for the estimation of steroids in blood may be unreliable and as only concentration is determined do not provide any indication of total hormone turnover

Several cases of active peptic ulceration in subjects with Addison s disease have been reported since cortisone therapy was introduced (Engel 1955) whereas the co-existence of the two diseases was an extreme rarity beforehand This suggests that the secretions of the adrenal cortex have a *permissive* role in the development of peptic ulcer and perhaps act by allowing normal metabolic activity in the parietal cells of the stomach with the production of the all important acid But a full understanding of the role of the adrenal cortex is complicated by the knowledge that steroids and A C T H can partially protect dogs from ulceration after short-circuit operations (Schien burger and Saltzstein 1954)

EVIDENCE FROM STUDIES OF GASTRIC SECRETION IN HUMANS

If histamine is infused intravenously at a constant rate into humans the acid secreting response ultimately reaches a level which remains constant (Fig 4) When a series of such observations are made on the same subject using different doses of histamine it is possible from the results to construct a dose response curve the points of which lie on a sigmoid shape and can be fitted to a logarithmic curve (Fig 4) The curve expresses the results of the interaction of histamine with acid secreting units of the stomach applying the simple mass action theory of physical chemistry By such application the maximal output of acid will be directly related to the total number of secreting units in the stomach (Adam *et al* 1954)

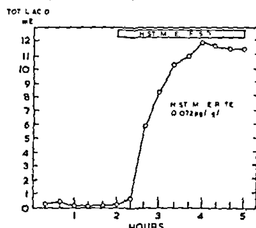
In man an association between acid secretion and the state of the gastric mucosa has been demonstrated (Surula and Lehtiner 1953) and Tongen (1950) found a correlation between the total acidity of stomachs before partial gastrectomy and the *concentration* of parietal cells in the resected portion of stomach Cox (1952) studied the size and weight of the stomachs removed at autopsy from subjects with chronic peptic ulcers and showed that there was a correlation between these factors and ulcer lesions The stomachs of subjects with duodenal ulcer were larger and heavier than normal while in subjects with gastric ulcer the stomachs were smaller and lighter Further

more there was a correlation between the volume of the mucosa and the number of parietal cells present in it The average number of parietal cells

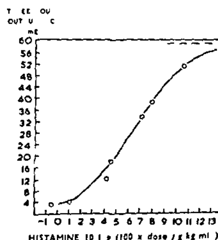
DOSE / RESPONSE CURVES FOR HISTAMINE
ON HUMAN ACID GASTRIC SECRETION

A om Card t 1954

If h tam ne is inf sed ntravenously at a constant rate the ac d secretory response ult mately reaches a constant le el



If a ser es of such obse vat ons a e m d on the same s bject at d fferent dose le els t is poss bl to construct a dose/re po se relat onsh p
The po nts lie on a s gmo d curve and ha e here bee f tted to a logar thm c c r e



Th s c e e p ess s the res lt of the i teract o of h stam e w th s cret g u ts of the stomach u g th simpl m ss ti theory of phy cal chemi try On thi th o y th ma mal o tp t of acid is d r ctly r l t d to th total numb r of s c t g u it in the st mach o th pa etal cell ma

Fig 4

in thirty cases of duodenal ulcer was 1 8 billion but was only 0 8 billion in twenty two cases of gastric ulcer These observations indicate the possible significance of the size of the parietal cell mass in relation to the aetiology

of peptic ulcer With the maximal' histamine dose response calculated as stated above and in relation to its role as an index of the parietal cell mass a tool is obtained for the true quantitative examination of both structure and function in the stomachs of subjects with peptic ulcer

To return to the concept of peptic ulceration resulting from the imbalance of forces of attack and defence several hypothetical situations productive of peptic ulceration may be conceived (Table II) The probability of an ulcer

TABLE II
HYPOTHETICAL SITUATIONS RESULTING IN PEPTIC ULCERATION

Situation	*Parietal Cell Mass Size*	*Neural Stimuli*	*Hormonal Stimuli*	*Defence Forces*	*Possible Result*
a	Normal	Normal	Normal	Normal	Healthy
b	Larger	Normal	Normal	Normal	D U
c	Larger	Normal	Normal	Inadequate	D U and G U
d	Normal	Normal	Normal	Inadequate	G U
e	Normal	Greater	Normal	Normal	D U
f	Normal	Normal	Greater	Normal	D U

It follows that any combination of these situations may occur to increase the risk of ulcer formation *e g* a large parietal cell mass may be associated with both inadequate defences and increased neural and hormonal stimuli

arising in an individual will depend upon which abnormal factor or combination of factors is operating As stated before knowledge of the variations in the forces of defence is wholly inadequate But the use of the histamine test has elucidated the importance of the forces of attack in the aetiology of peptic ulcer Gastric and duodenal ulcers may be considered together for the nature and behaviour of the lesions are identical The difference in the associated features of secretion and motility can be comprehended as a reflection of variation in the imbalance of forces represented in Table II Indeed the two lesions frequently coexist Of 220 duodenal ulcers in males consecutively admitted to the Edinburgh Unit in the past three years forty four or exactly twenty per cent had an associated active gastric ulcer Put in another way of eighty four cases of gastric ulcers in males studied during the same period forty four had a coexistent duodenal ulcer (52 3 per cent) The natural history in these cases indicated that in the majority the duodenal ulcer preceded the gastric ulcer by some years Some alteration in the type of forces active in the various situations outlined in Table II would explain this sequence *e g*

from situation b to c The singular difference in the sex incidence of this association of gastric and duodenal ulcers throws *some light on aetiology of* peptic ulceration Of sixty five cases of duodenal ulcer in females the co-existence of a gastric ulcer was encountered only four times (6 2 per cent) of forty four cases of gastric ulcer in females a duodenal ulcer was associated in only 9 1 per cent If it is assumed that in the balance of forces inadequate defences are more likely to be responsible for the development of gastric ulcers this clear difference in sex incidence suggests that hormonal influences may be important in defence of the mucosa against digestion

RELATIONSHIP OF ACID POTENTIAL TO PARIETAL CELL POPULATION IN RESECTED PORTION OF STOMACH

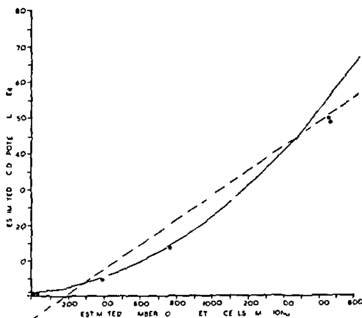


FIG 5

THE RELATION OF PARIETAL CELL MASS TO ACID OUTPUT

Although there is at present no means of measuring the parietal cell mass of the intact stomach it is possible to make parietal cell counts from the removed portion of stomach after a partial gastrectomy The maximal output of acid corresponding to these cells can be determined by measuring that from the intact stomach before operation and subtracting the output obtained from the stomach remaining after operation (Marks 1957) By examining in this way a series of stomachs (Fig 5) it is possible to establish

a strong correlation between the two variables parietal cell mass and the estimated acid output of the resected part of stomach (Cards and Marks 1959) It appears therefore that under the conditions of the maximal histamine test the acid response is proportional to the numbers of parietal cells present and that low outputs are associated with few cells large with many and since the curve runs through the origin no acid with an absence of cells Thus in the absence of artificially exaggerated neural and hormonal influences the results of the maximal histamine test give direct information on the size of the parietal cell mass The advantages of this test of gastric secretion over previous methods lies in its quantitative nature and its repeat ability tests done months apart on the same subjects have shown minimal variation and often remarkable reproduction—the coefficient of variation in thirty seven tests on fifteen subjects repeated at intervals up to two years was nine per cent The application of this test to the study of peptic ulcer (Bruce *et al* 1959) has provided the results detailed in Table III

If the hypothesis of the relation of acid output to parietal cell mass is correct the data in Table III suggest that duodenal ulcer arises in association with a greater than normal number of parietal cells and gastric ulcer except where complicating a duodenal ulcer does not have this association It is conceivable that the low output of acid in gastric ulcer is due either to local vascular and metabolic disturbance arising from the proximity of the ulcerative lesion to the parietal cell tissue or possibly to the primary general disturbance of the stomach which affecting the defence mechanisms permits digestion of the mucosa

There is still no good evidence of when the abnormal secretory activity associated with peptic ulcer first develops and if it precedes the ulcer by how long A follow up of 100 medical students who had had a histamine augmented fractional test meal fifteen years before (Doll *et al* 1949) showed that ten out of the traceable eighty five subjects had developed peptic ulcer during the interim period All ten of these subjects with ulcer had originally been in the group which secreted the higher amounts of acid in response to the fractional test meal

In a study of the nature of the hypersecretion in duodenal ulcer (Hunt and Kay 1954) it was concluded that the percentage of maximum secretory capacity put out under basal conditions by subjects with duodenal ulcer was not different from that in normals and it was assumed that the higher basal secretion in duodenal ulcer must be accounted for simply by the greater number of parietal cells in the stomach The results using the histamine test (Table III) do not wholly confirm the observations of these workers Calculated from the means the normal ratio of maximal to basal output is approximately 10 1 for both sexes But in duodenal ulcer subjects it is 6 1 for males

and 7 1 for females Furthermore in males with duodenal ulcer when the basal output is less than 4 mEq (eighty nine cases) the ratio is the same as

TABLE III
SECRETORY RESPONSE TO MAXIMAL HISTAMINE STIMULATION*

Group	*No of Subjects*	*Basal Secretion mEq HCl*		*Maximal Secretion mEq HCl*	
		Mean	*Range*	*Mean*	*Range*
Normal males	14	2 5	0 9 5	22 4	10 1 34 6
Normal females	18	1 3	0 6 8	14 6	0 1 31 3
Duodenal ulcer					
(*a*) Uncomplicated					
males	176	6 0	0 1 23 1	37 5	4 2 91 7
females	61	3 2	0 1 14 9	24 3	4 6 43 9
(*b*) Pyloric stenosis					
males	22	5 8	0 8 22 1	37 8	13 7 59 8
Concurrent duodenal and gastric ulcers					
males	44	4 7	0 3 23 0	30 1	0 6 88 0
females	4	1 3	0 0 3 7	15 8	0 2 31 6
Gastric ulcer					
Uncomplicated					
males	40	2 1	0 1 14 4	14 6	1 7 45 9
females	40	1 2	0 0 7 9	12 7	0 5 40 8
Post operative dyspepsias					
(*a*) Recurrent gastric and suture line ulcers	9	1 4	0 15 4 2	4 3	0 8 14 7
(*b*) Jejunal ulcers—					
following gastro enterostomy	10	8 7	5 1 30 0	43 5	26 8 60 7
following gastrec tomy	14	7 7	0 6 19 8	31 1	7 7 82 0
(*c*) Dyspepsia but no ulcer	8	1 5	0 1 4 0	4 7	0 3 11 3

* *The Maximal Histamine Test* The patient fasts overnight A radio opaque Levine tube is passed and the tip screened into position in the antrum The resting juice is aspirated For one hour the patient lies on the left side and basal secretion is aspirated continuously with an electric pump After 40 minutes the anti histamine mepyramine 50 mg is injected I M and at the end of the basal hour histamine acid phosphate 0 04 mg per kg /B W of patient is injected subcutaneously Continuous aspiration is maintained thereafter for one hour Basal secretion refers to that aspirated in the hour before the injection of histamine and maximal to that in the subsequent hour

in normals falls to 5 7 1 in the cases with a basal output of 5 9 mEq (fifty five cases) and to 3 8 1 in the twenty three subjects with a basal output over 10 mEq Thus in half of the subjects with duodenal ulcer the stomach at rest secretes in the absence of exogenous stimuli at a rate higher than

can be accounted for solely on the basis of the number of secretory units Put in terms of the percentage of maximal capacity to secrete occupied by the basal output the results are for the eighty nine cases with a basal secretion under 4 mEq /hour 8 6 per cent for the fifty five cases with a basal output of 5 9 mEq /hour 17 5 per cent and for the twenty three cases secreting more than 10 mEq in the basal state, 26 3 per cent These figures are to be compared with the mean percentage in normals of 11 1 per cent In the

TABLE IV
THE RELATION OF THE RESTING SECRETION TO THE HISTAMINE STIMULATED SECRETION IN DUODENAL ULCER IN MALES

Output in Basal Hour	*No of Cases*	*Mean of Output in Post Histamine Hour*	*Ratio of Mean Maximals to Mean Basal Outputs*
Over 10 mEq	22	49 03 mEq	3 8 1
5 9 mEq	55	40 05 mEq	5 7 1
0 4 mEq	89	30 58 mEq	11 5 1

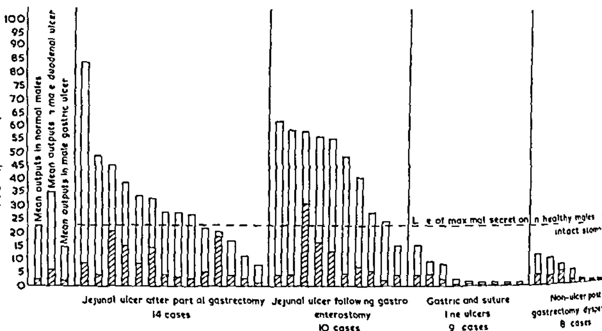
TABLE V
THE RELATION OF THE ACID OUTPUT TO THE DURATION OF HISTORY IN DUODENAL ULCER IN MALES

Length of History in Years	*No of Cases*	*Output of HCl in mEq in Basal Hour*	*Output of HCl in mEq in Post Histamine Hour*
0 1	11	3 65	29 4
2 7	46	4 70	33 8
8 13	34	5 97	37 0
14 19	20	5 30	39 6
Over 20	38	5 66	35 9

resting state therefore in half of the subjects with duodenal ulcer either stimuli are operating to keep a larger number of secreting units actively at work than in normals or all the secreting units are putting out acid at a greater rate On the other hand whereas in duodenal ulcer subjects there is direct correlation between basal and maximum secretions this does not appear in normal subjects where the basal output shows unmatched variation (Bruce *et al* 1959)

A consideration of the output and the duration of the history in duodenal ulcer in males reveals a significant positive relation for both basal and maximal figures with an upward trend in secretion for histories up to twenty years (Table V) Thereafter the relation disappears but the operation of senescence and of degenerative changes secondary to inflammation in the gastric mucosa

could reasonably be held to account for this change after twenty years This observation suggests the possibility that hyperplasia of the parietal cell mass occurs with the chronic operation of secretion exciting stimuli



THE ACID OUTPUT IN THE BASAL AND POST-HISTAMINE HOURS IN CASES OF RECURRENT DYSPEPSIA FOLLOWING GASTRIC OPERATIONS

FIG 6

THE ACID OUTPUT IN POST OPERATIVE ULCER RECURRENCE

The maximal histamine test can be applied effectively to the study of recurrent ulceration following gastric operations (Marks 1957) Recent observations (Bruce *et al* 1959) are summarised in Table III and Figure 6 In all of ten subjects under investigation for disability following partial gastrectomy in whom no evidence of recurrence of ulcer was disclosed the maximum output was less than 10 mEq or less than half that of the normal intact stomach In fourteen subjects with proven jejunal ulcer after partial gastrectomy the mean output was 31 1 mEq and the basal 7 7 mEq *i e* the output of the gastric remnant was greater than that expected from a normal intact stomach and only a little less than that found to be the average for the intact stomach in cases of duodenal ulceration In this group of recurrent ulcers the mean basal secretion was actually higher than that for the group of uncomplicated duodenal ulcers in males Furthermore the ratio of basal to maximum 1 4 resembles the results in the major secreting group of duodenal ulcers and suggests that it is from those of this group undergoing

operation that the recurrent jejunal ulcers are produced This is also concluded from the observation that in ten subjects with recurrent jejunal ulcer following gastro-enterostomy the mean maximal output of 43 5 mEq and the basal of 8 7 mEq is higher than that of uncomplicated duodenal ulcer in males Hence in this group of post-operative subjects whose recurrent ulcers resemble experimentally produced ulcers in animals the direct relation between acid and ulcer is quite clear Nine other subjects had recurrence of ulceration not in the jejunum but in the stomach remnant or on the suture line The mean maximum output in these subjects was only 4 3 mEq Thus the recurrence of ulcer in these sites is associated with a low production of acid and emphasises the greater importance of local nutritional or ischaemic changes in the aetiology of gastric ulcer

THE ASSOCIATION OF PEPTIC ULCERS WITH EXTRA GASTRIC TUMOURS

The association of peptic ulceration with tumours outside the stomach and duodenum has recently excited considerable interest One association is with metastatic carcinoid tumours in thirty-eight per cent of which cases a peptic ulcer has been found (Macdonald 1956) Why this should be is not clear 5 Hydroxytryptamine is known to have the property of inhibiting acid gastric secretion and while some tumours may be histamine producing or histamine releasing increased output of acid has not been demonstrated in these cases Another group associates peptic ulcer with hyperparathyroidism usually due to parathyroid adenomata The incidence of the complication varies from fifteen to thirty per cent in the reported series Again it is not known why this relationship occurs Excess calcium in the blood tends to depress gastric secretion and although experimentally a deposition of calcium in the fundic glands can be demonstrated no peptic ulceration results That an abnormal gene may be involved is suggested by the tendency in this relationship of peptic ulcer to adenomata for such tumours to be multiple The association of hyperparathyroidism and peptic ulcer has recently been reviewed (Kirsner 1958) A third and most interesting if rare group of ulcers was first given publicity by Zollinger and Ellison (1955) This group consists of young subjects with persistent ulceration of stomach and duo denum often with multiple episodes of perforation and bleeding Investiga tion discloses a remarkable output of acid throughout the day and night and abdominal exploration reveals a tumour of the pancreas and sometimes other tumours as well Islet cell tumours with secreting or non secreting beta cells or alpha cells or mixed forms have been described Most of these subjects ultimately have to suffer total gastrectomy such is the output of acid from

even the smallest gastric remnant There are two possible explanations for such cases The first is that the high acid secretion is the result of hyperinsulin hypoglycaemic stimulation of the vagus but in fact no effective hypoglycaemia has been demonstrated in these cases In this connection it is of interest to note that Platt *et al* (1949) failed to demonstrate any evidence of different reactive blood sugar levels between duodenal ulcer cases and controls given oral and intravenous glucose The second possibility is that an ulcerogenic factor might be secreted by the pancreas and that it may be glucagon This possibility is supported by the observation that duodenal ulceration follows the experimental diversion of pancreatic secretions from the duodenum (Matthews and Dragstedt 1932) while it rarely follows total pancreatectomy (Berg 1934) But examination of the serum of subjects with persistent ulceration has not revealed any abnormal glucagon activity and intravenous administration of glucagon has not consistently affected gastric secretion and did not cause ulceration when administered over thirty days to dogs (Elrich H quoted by Eisemen and Maynard 1956) Another possibility is that in such subjects the prolonged stimulation of acid may result from constant activation of the mechanisms responsible for the delayed response to insulin described above A case is reported in which a duodenal ulcer developed in 1942 perforated in 1943 had repeated bleeds thereafter to 1947 and jejunal ulcer two months after a three quarters gastric resection A gastric ulcer developed seven years after subsequent vagotomy and a further resection left only one tenth of the stomach but again a jejunal ulcer appeared after that (Eiseman and Maynard 1956) At final total gastrectomy a mixed alpha and beta cell tumour of the pancreas was found This subject had had adenomas of parathyroid and pituitary and an insulinoma dealt with in previous years However in the Edinburgh Gastro-intestinal Unit we have studied a man with a similar saga of recurrent haemorrhages perforations and ulcers in whom repeated search has failed to disclose any evidence of such adenomata The tiny gastric remnant left after three partial gastrectomies and a vagotomy has produced as much acid in response to the histamine test as would be obtained from an intact stomach While therefore the parietal cell population in such subjects must be immense there is likely to be another factor producing a tremendously augmented output of acid from the secreting units If in such grotesque cases the mechanism can be disclosed it may provide clues for the aetiology of their less florid cousins

Summary

It has been shown that multiple factors are involved in the development of a peptic ulcer and that the essential common result is the upsetting of the balance of the forces of attack and defence Increased power to digest

the result of unusual size of the parietal cell mass or of an unusual activity of the cells or inadequacy in the forces of defence because of structural degeneration or interference with cell metabolism may equally result in digestion of the alimentary mucosa

Genetic neural and hormonal mechanisms may be responsible for increase in the forces of attack and metabolic hormonal and vascular disturbances for decrease in the powers of defence The natural resistance of the alimentary mucosa to digestion varies throughout the tract and situations wherein parts with poor natural resistance are exposed to even subnormal quantities of acid and pepsin are conducive to peptic ulceration

Advances in our knowledge of these various mechanisms should eventually lead to rational management with attention being directed to those particular aetiological factors which are at work in the individual whose peptic ulcer is being treated

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CHAPTER III

PATHOLOGY OF CHRONIC PEPTIC ULCER

By R WINSTON EVANS

IT is not the intention of the writer to discuss the pathology of chronic peptic ulcer extensively *this has been done already by several authors* notably Stewart (1929 1953) Herein only the main features will be mentioned concisely

Site and incidence

Although the vast majority of peptic ulcers occur in the stomach and duodenum lesions of a similar character infrequently develop in the distal portion of the oesophagus in the jejunum (after gastro-enterostomy) and more rarely in other parts of the small intestine In a Meckel s diverticulum the mucosa adjacent to the heterotopic gastric epithelium also may be affected by peptic ulceration

In the stomach peptic ulcers exhibit a marked tendency to be restricted to an ulcer bearing area which includes the lesser curvature and the adjacent anterior and posterior surfaces from the cardio-oesophageal junction to the juxta pyloric region Indeed about 75 per cent of chronic benign gastric ulcers are located in a segment which extends one inch on either side of the lesser curvature proximally for a distance of about five inches from the pyloric channel (which is defined as the narrow channel extending proximally for three quarters of an inch from the microscopically delineated gastro-duodenal junction (Foulk *et al* 1957))

The reported incidence of benign ulcers in the pyloric part of the stomach varies considerably This variation is accounted for partly by the confused and unsatisfactory *nomenclature for the different regions of the* stomach and partly by the source and nature of the material investigated Clinically twenty six to thirty five per cent of benign ulcers occur in the pyloric region *i e* at or distal to the incisura angularis (Swynnerton and Tanner 1953 Smith *et al* 1953) In post mortem specimens however only twelve per cent of gastric ulcers are located in this part of the stomach (Hurst and Stewart 1929) An ulcer may extend from the stomach or from the duodenum into the pyloric channel Most so-called juxta pyloric ulcers are in fact situated in the duodenum

In men benign gastric ulcers are distributed in a relatively even pattern from the cardia to the pylorus there being only a slightly increased incidence

in the middle and lower portions of the body of the stomach In women however seventy five per cent of gastric ulcers develop in the upper and middle parts of the body (Swynnerton and Tanner 1953) Five per cent of all gastric ulcers are situated on the lesser curvature within two inches of the cardia (Braasch *et al* 1955)

Ulcers in the fundus of the stomach *i e* above the level of the cardio-oesophageal junction and along the greater curvature are very uncommon occurring in only 1 5 per cent and three per cent respectively of all gastric ulcer cases (Welch and Burke 1958) It was formerly believed that the majority of ulcers in the pyloric region and along the greater curvature were malignant This is wrong since of all ulcers found in these situations (excluding lesions obviously malignant when first seen) over eighty per cent are benign on pathological examination (Marshall 1953 Bernardo *et al* 1958 Welch and Burke 1958)

In the duodenum the ulcer bearing area usually is the anterior or posterior wall of the first part an area extending for three quarters of an inch to an inch and a quarter from the pylorus More than seventy five per cent of duodenal ulcers are situated in this zone the remainder developing in the second part of this viscus It is rare for an ulcer to develop beyond the papilla of the main pancreatic duct

From post mortem studies Stewart (1929) found that chronic duodenal ulcer occurred nearly twice as commonly as benign gastric ulcer Statistics based on surgical considerations however show that the incidence of duodenal ulcer is more than twice that of gastric ulcer Of 1 285 cases of ulcer collected from three Liverpool hospitals and diagnosed by macroscopical criteria 860 were situated in the duodenum and 377 in the stomach (Clarke *et al* 1955) duodenal ulcer occurred eight times and gastric ulcer three times more frequently in males than in females Apart from gastric ulcer in women all types of peptic ulcer are commoner in the northern half than in the southern half of Britain (Watkinson 1958)

MULTIPLE ULCERS —In the Liverpool series of peptic ulcers (Clarke *et al* 1955) concomitant ulcers in the stomach and duodenum were noted in only 3 7 per cent of the cases An incidence as high as 14 4 per cent has been reported for these double lesions (Wilkie 1926) but a more representative figure is the nine per cent reported by Johnston (1955) who believes that the duodenal ulcer is present first and that the stomach ulcer develops secondarily as the result of gastric stasis In a series of 313 patients with gastric ulcers reported by Aagaard *et al* (1959) the disease began with a duodenal ulcer in 30 per cent in only 6 per cent did a duodenal lesion appear after the gastric ulcer The presence of an ulcer in the duodenum does

not necessarily mean that a concomitant ulcer in the stomach is benign in six per cent of malignant gastric ulcers there is a duodenal ulcer as well (Bernardo *et al* 1958)

Benign gastric ulcers are multiple in approximately twenty per cent of cases but it is much less common to find multiple lesions with malignant gastric ulcers (Dolphin *et al* 1953 Bernardo *et al* 1958) Sometimes multiple acute erosions develop in the stomach when there is an established duodenal ulcer and similar satellite lesions can develop in the mucosa surrounding a benign gastric ulcer In the duodenum twin as well as kissing ulcers are relatively common

Macroscopic features

A gastric ulcer presents a characteristic appearance it is customarily round or oval regular and sharply defined occasionally it is saddle shaped The crater is surrounded by smooth mucosa which approaches or may overhang the excavated portion so that the ulcer edge is rolled or sloped or rolled at one point and sloped at another but never shelved like a malignant ulcer Ordinarily very little heaping up of the margins is seen a characteristic helpful in distinguishing a benign gastric ulcer from an ulcerated carcinoma in which the margins are irregular and nodular Occasionally however such a differentiation cannot readily be made from the consideration of the naked eye features alone and as an early carcinoma infrequently may suffer a peptic type of ulceration it is important not only to select several portions of the ulcer edge but also as many neighbouring lymph nodes as can be found for histologcial examination Frequently a chronic atrophic gastritis surrounds the ulcer (Magnus 1952)

The walls of a benign ulcer descend either vertically to impart a punched out appearance or steeply in a somewhat terraced fashion with the proximal side inclining towards the floor Although commonly there is no undermining the breach in the muscularis propria usually is so complete that the severed muscle fibres retract to produce a gap in the deep part of the crater which is wider than that in the mucosa Occasionally the mucosal hiatus is insignificant only one tenth inch in diameter yet the ulcer penetrates deeply Characteristically in resected specimens the lesion does not exceed one inch in diameter but occasionally a benign ulcer exceeds two inches in diameter and may be saddle shaped straddling the upper part of the lesser curvature

When the lesion has extended to the subserous layer this surface becomes thickened and oedematous fibrous sometimes puckered and often speckled by numerous congested capillary vessels Tags of omentum fasten to the affected serosa and adhesions may develop binding this surface firmly to adjacent organs

The gross morbid anatomy of duodenal ulcers is essentially similar to that of gastric ulcers A duodenal ulcer is round or oval and seldom attains a large size Since most posterior ulcers penetrate into the pancreas and many of the less common anterior ones perforate in resected specimens the ulcer is usually damaged and incomplete its floor being absent Commonly the layers of an intact duodenal ulcer are thinner and less indurated than those of a gastric ulcer



FIG 7
Duodenal ulcer in a male aged 40 years there was a concomitant gastric ulcer The four characteristic layers are shown and the lesion spreads to involve and almost perforate the congested serosa H & E × 30

Microscopical appearances

The margins and floor of a peptic ulcer exhibit the characteristic features of a chronic inflammatory process in which zones of exudation of necrosis of granulation tissue and of fibrosis can be distinguished (Fig 7) The ulcer crater is lined by an inner layer of leucocytes enmeshed in fibrin which is easily washed away Beneath this is a zone varying in thickness of fibrinoid necrosis which in turn is firmly fixed to an underlying cushion of granulation tissue (Fig 8) The deepest layer composed of scar tissue extends laterally for some distance beyond the mucosal limits of the lesion and forms the base of the ulcer When the lesion has eroded completely through the muscularis propria the base of the ulcer may be composed of the parenchyma of a contiguous organ

In its active phase a peptic ulcer is infiltrated by polymorphonuclear leucocytes but collections of eosinophils lymphocytes and plasma cells are more characteristic of the markedly indurated chronic lesion In the latter type of lesion the eosinophils are chiefly situated in the fibrinoid necrotic layer and the lymphocytes and plasma cells lie in groups mainly among and around the numerous vessels of the zone of granulation tissue



FIG 8

Same tissue as previous figure showing leucofibrinous and necrotic layers with underlying granulation tissue Section through floor of ulcer H & E ×115

There is a tendency for the muscle fibres at the edge of a benign gastric ulcer to spread out fanwise (Fig 9) and to fuse with the fibres of the muscularis mucosae (Newcomb 1932) The significance of this microscopical feature will be discussed later in connection with ulcer-cancer

At the margins of a peptic ulcer especially of an active lesion the epithelium sometimes exhibits evidence of proliferative activity and is frequently distorted by underlying granulation and fibrous tissue The tubules of the mucosa become dilated assume irregular shapes and appear to be

growing down below the muscularis mucosae into the deeper tissues It is therefore important that this histological appearance particularly the position of the atypical epithelial structures should not be interpreted as evidence of malignancy

The gastritis present in association with peptic ulceration is of a non specific character Sometimes there is a marked degree of gland destruction and a dense cellular infiltration Commonly metaplastic areas of intestinal epithelium are found in association with gastric and duodenal ulcers This



Fig 9
Splayed out muscularis mucosae at the edge of an ulcer cancer H & E ×65

intestinal metaplasia (Fig 10) renders the affected stomach in the opinion of Morson (1955) more prone than the normal to the development of carcinoma The metaplasia is sometimes diffuse but more often is confined to the pyloric region where columnar cells goblet cells and Paneth cells are occasionally found forming tubular structures resembling the intestinal glands of Lieberkuhn The precise relationship however of metaplasia to either peptic ulcer or carcinoma has at present not been evaluated

The healing of a peptic ulcer is accomplished by the organisation of granulation tissue and epithelialisation of the mucosal hiatus New glands are not formed the muscle is not regenerated and the scar that remains is covered only by a single layer of epithelium

Blood vessels in a peptic ulcer —The stomach is one of the most vascular organs in the body In the submucous layer there is a very large and complicated network of blood vessels from which smaller vessels pass

inwards to form secondary networks in the mucosa The submucous layer also contains numerous arterio venous shunts (Barclay and Bentley 1949) but the mechanisms controlling these shunts are still unknown



FIG 10
Epithelium of intestinal type with many goblet cells at edge of a malignant peptic ulcer in a male aged 54 years H & E × 115

When a peptic ulcer penetrates into the wall of the stomach or duodenum the changes in the blood vessels depend on how fast the ulcerative process is proceeding With an acute ulcer the wall of an artery may be eroded before there is sufficient time for reactive changes to appear Since the artery is essentially normal and since the opening in it is often a lateral one this type of erosion causes brisk haemorrhage (Osborn 1954) When the ulcer is developing more slowly a partially organised thrombus is usually found in the lumen of any vessel that is eroded In a chronic ulcer the intima of the vessels proliferates and becomes infiltrated with mucinous material to such an extent that the lumen is blocked This prevents any serious haemorrhage when the vessel is eroded but by reducing the blood supply interferes with healing

NERVES IN A PEPTIC ULCER —Nerves as well as arteries suffer injury as the ulcerative process extends At the edge of the ulcer fragmented nerves lie embedded in the inflammatory tissue (Fig 11) Such affected nerves are drawn into this position often under cover of an intact but thickened mucosa or submucosa by the retracting severed muscle layers (Kinsella 1953) Peri neural inflammation and consequent fibrosis are observed commonly the



FIG 11
Hypertrophied nerve undergoing changes due to early involvement by inflammatory cells This is from a zone immediately adjacent to the ulcer crater H & E ×105

ganglion cells disappear and the disintegrating nerves become surrounded by masses of small round cells Sometimes the affected nerve fibres undergo disordered regenerative changes producing end bulbs comparable with traumatic neuromata Not only at the edge of an ulcer but also in its base nerve fibres are found and occasionally these appear much larger than normal and are in various stages of hypertrophy and degeneration These neural changes are of interest especially in relation to the pain of peptic ulceration

Penetration

Penetration beyond the wall of the alimentary tract into an adjacent viscus or structure is a common complication of peptic ulceration It occurs more frequently than free perforation into the peritoneal cavity (Haubrich *et al* 1953) Its exact incidence is much greater in surgical series than in post mortem studies because penetration is the outcome of a severe type of

ulceration which is rather resistant to medical treatment In a series of cases treated by subtotal gastrectomy penetration had occurred in almost thirty per cent of duodenal and of jejunal ulcers but in only eighteen per cent of gastric ulcers (Haubrich *et al* 1953) Firm fibrous adhesions (without actual penetration) were present in similar percentages in the three types of ulcer

In two thirds of cases penetration takes place into the pancreas Fortunately the main pancreatic duct mostly escapes damage and an internal pancreatic fistula is very uncommon (Lavery and Kyle 1959) Adhesions to the biliary tract are present in approximately one third of surgical cases Other structures and viscera are involved much less frequently by peptic ulceration but an ulcer can rarely penetrate into almost any structure in the upper abdomen and lower thorax In civilised countries it is now very rare for a primary ulcer to involve the anterior abdominal wall but a jejunal ulcer—especially after an ante-colic anastomosis—can do so (Foster and Carlson 1958) Exceptionally an ulcer penetrates upwards into the heart (Johannessen 1946) or pleural cavity (Hudson *et al* 1945) forwards into the colon (Starzl *et al* 1959) or backwards into the renal pelvis (Stock 1954) Slightly more common is the establishment of a fistula usually from the duodenum to the gall bladder or common bile duct (Hutchings *et al* 1956 Kyle 1958) A patient with such a fistula presents with either recurrent cholangitis or with pyloric obstruction caused by the inflammatory mass

Cicatricial contracture

Chronic duodenal and prepyloric ulcers can lead to organic pyloric stenosis caused by the contraction of fibrous tissue The size of the lumen is frequently still further reduced by superadded spasm inflammation and oedema The stenosis is sometimes accompanied by marked hypertrophy of the pyloric musculature and the gastric mucosa although inflamed is still capable of secreting large quantities of acid The contracting fibrous tissue not only causes a deformity to develop by shortening and then fixing the proximal part of the duodenum but also results in the production of single or multiple small pseudo-diverticula situated between the ulcer and the pylorus on occasions it distorts dangerously the normal anatomy of such important structures as the bile and pancreatic ducts

In the stomach a chronic ulcer especially if saddle shaped on the lesser curvature causes the greater curvature to be gradually indrawn so that the stomach is to a varying degree divided into two sections Such an hour glass deformity is now very rare in Northern England and Scotland and is of more historical than practical interest It is still seen occasionally in Southern England mostly in elderly women Hour glass deformity occurs also in

carcinoma of the stomach and is the commoner cause of this appearance *certainly* in men, and possibly today in women as well

Post operative gastritis and post operative gastric cancer

Of considerable interest to the surgeon operating on patients for peptic ulcer disease is the possibility that a carcinoma may develop years later in the gastric stump (Krause 1957 Pack and Banner 1958) This is surprising in view of the fact that the chief cancer bearing area is removed during gastrectomy Such a predisposition to develop gastric cancer in the remaining portion of the stomach seems valid from the analysis of Helsingen and Hillestad (1956) who included in their series only those persons remaining alive for more than five years after operation These authors found that in the group operated on for gastric ulcer the observed frequency of gastric cancer developing in the gastric stump was approximately three times as high as expected In the group operated for duodenal ulcer no statistically significant proclivity to develop cancer could be demonstrated Superficial gastritis is common after operations on the stomach especially when a gastrojejunal anastomosis has been made (Palmer 1953) and there may be marked intestinal metaplasia of the gastric mucosa These changes are presumably associated with the reflux of duodenal contents into the stomach Whether or not such contents and the concomitant gastritis can act as promotional agents in the genesis of cancer is not known

Recurrent ulceration

The subject of recurrent ulceration following gastric surgery is dealt with fully in Chapter XIII Recurrent ulcers usually develop at the stoma or in the distal bowel within two inches of it Less commonly they can appear in the stomach and rarely after a gastro jejunal anastomosis in the afferent loop The microscopical features and the complications of recurrent ulcers are similar to those of gastric and duodenal ulcers The sinister reputation which they have acquired is due to their proximity to important structures such as the transverse colon and middle colic artery and to the fact that they are the product of a severe ulcerogenic tendency which the surgeon has failed to control

Associated diseases

Reference has already been made in the preceding chapter to the association between peptic ulceration and (1) parathyroid tumours (2) metastasising carcinoid tumours of the small bowel and (3) non insulin secreting and less commonly functioning islet-cell lesions in the pancreas (Cunningham *et al*

1952) The lesions in the pancreas take the form of benign or frequently malignant neoplasms of the islet cells or rarely simple islet-cell hyperplasia (Zollinger and McPherson 1958 Summerskill 1959) The staining reactions of the neoplastic cells are sometimes those of typical β-cells (Evans 1956) in functioning islet-cell tumours associated with peptic ulceration In non functioning tumours occurring together with peptic ulcer however the demonstration of β-cells as in the case described by Strøm (1952) does not appear to be of significance alpha granules are sometimes seen but tumours composed of cells containing beta granules are rare (Donaldson *et al* 1957) The exact relationships between the different types of islet cells their secretions and the syndromes which they can produce are still obscure Along with the pancreatic lesions there are sometimes multiple adenomata in other endocrine glands especially the parathyroids pituitary and adrenals (Fisher and Flandreau 1957 Zubrod *et al* 1958) The peptic ulceration present in these cases is of a very severe type There are usually multiple ulcers in the distal part of the duodenum or proximal jejunum and the discovery of such ulcers should always suggest the possibility of an islet-cell tumour Recurrence after conventional types of gastric surgery is rapid and perforation common The ulceration is often accompanied by severe diarrhoea or steatorrhoea (Priest and Alexander 1957 Summerskill 1959) indeed diarrhoea can be the only gastro-intestinal manifestation of the islet-cell abnormality (Verner and Morrison 1958)

Tumours of the islet cells which secrete insulin and lower the blood sugar are only infrequently associated with peptic ulceration (Janowitz and Crohn 1951 MacGregor 1958) In one recently reported case (Pender 1959) a familial factor was apparent A father with recurrent peptic ulceration and who had been receiving psychiatric treatment was found to have a beta cell tumour of his pancreas at the same time a similar type of tumour which had been causing the typical symptoms of hyperinsulinism was removed from his daughter

In cirrhosis of the liver peptic ulceration is not uncommon the reported incidence varying from four to twenty five per cent (Koide *et al* 1958) Unfortunately if portal hypertension develops and a porto-caval shunt is performed a gastric ulcer is likely to develop (Clarke *et al* 1958) These post shunt ulcers may be due to some intestinal secretagogue not being destroyed in the liver

Crohn s disease and duodenal ulceration both occur in patients of similar age but the existence of duodenal ulceration in from six to twenty three per cent of patients with Crohn s disease (Freud and Spellberg 1957 Pollock 1958) represents a concurrence rate greater than can be explained by the similar age distribution of the two diseases Peptic ulcers develop in eight

per cent of patients with polycythaemia vera (Wilbur and Ochsner 1935) and this association too has never been satisfactorily evaluated or explained

Hiatus hernia and peptic ulcer often co-exist A duodenal ulcer is present in up to twenty per cent of cases (Carver 1958) and frequently an ulcer is situated in the hiatal region With a sliding hernia an ulcer can exist either in the part of the stomach which has herniated up into the chest or on the posterior wall of the oesophagus which is exposed to acid pepsin digestion (Barrett 1950) With a rolling or para oesophageal hernia it is in the fundus of the stomach usually where it is stretched across the left crus of the diaphragm (Davidson 1958)

Acute peptic ulceration is present thirteen times more frequently in patients dying of emphysema than in other types of case coming to necropsy (Flint and Warrack 1958) Apart from its association with smoking chronic bronchitis alone does not appear to have any special significance in peptic ulcer disease (Allibone and Flint 1958) Coronary thrombosis and ulcer are however rather more commonly associated than can be accounted for by chance (Watkinson 1956)

It has been known for a long time that certain lesions of the brain can cause acute peptic ulcers The lesions most likely to do so are situated in the midbrain and hypothalamus (French *et al* 1952 Watson and Netsky 1954) and the ulcers are nearly always of the acute type A severe burn often causes a similar type of ulcer—the so-called Curling s ulcer first described by Long of Liverpool in 1840 Such ulcers are present in about twenty per cent of fatal cases of burns (Hummel *et al* 1957) It is now recognised that they may be comparable with the ulcers which sometimes develop after a wide variety of diseases injuries and operations (Fletcher and Harkins 1954 Roberts 1954) and they are currently attributable to the results of the patients response to stress Changes in the adrenals are found in thirteen per cent of ulcer patients studied at post mortem (Ellison *et al* 1959)

'CARCINOMA EX ULCERE'

Not only primary carcinomata of the stomach but also other neoplasms arising primarily in this viscus such as leiomyomata leiomyosarcomata and neurofibromata may suffer ulceration Some primary gastric carcinomata however appear to be ulcerated lesions from their inception and the carcinomatous tissue in the edges and bases of their craters is accompanied by a marked fibroblastic reaction Such lesions unless examined carefully may be occasionally mistaken for peptic ulcers or else wrongly interpreted as carcinoma ex ulcere

When carcinoma develops in a pre-existing benign ulcer the crater usually appears deep and punched out not shallow as in an ulcerated cancer and its mucosal margins overhang the edges which are not shelved and irregularly nodular as in the ulcer malignant *ab initio* As mentioned above a gastric carcinoma may undergo peptic digestion and ulceration and in this third *type of lesion the superficial cancer may be restricted to an interrupted narrow* often fragmentary ring like peripheral zone in the edges and overhanging margins surrounding a deep crater lacking neoplastic elements in its base Rarely the erosion is so complete that the diagnosis of malignancy is established with certainty only by the demonstration of metastatic deposits in the subjacent lymph nodes

Although many writers including Stewart (1929 1947) and Harnett (1947) consider that no more than eleven per cent of peptic ulcers are liable to become malignant the relationship of chronic benign gastric ulceration to cancer of the stomach remains debatable Indeed the incidence of ulcer cancer as assessed by various observers lies at different points within the range of one to seventy-one per cent Whereas Marshall (1953) and Swynnerton and Truelove (1951) believe it to be improbable that more than five to seven per cent of gastric carcinomata originate in pre-existing peptic ulcers Newcomb (1932) is of the opinion that approximately thirteen per cent of gastric cancers are carcinoma ex ulcere Stewart s last assessment (1953) of gastric carcinoma arising in antecedent peptic ulcer approaches twenty per cent

Any opinion expressed on the origin of gastric cancer in a benign chronic ulcer is largely dependent on the interpretation by the pathologist of the relevant macroscopic and microscopic features of such lesions Provided that the ulcerated cancer is large and deep and contains in its base enough scar tissue to suggest that it has been preceded by a chronic lesion of considerable duration the lesion is often considered to be an instance of carcinoma ex ulcere The age of a peptic ulcer however together with the nature of the histological features on which its remnants can be recognised in an ulcerated cancer are still subjects of much controversy Newcomb (1932) reviewed the microscopic features which should be demonstrated before a diagnosis of carcinoma ex ulcere could be confirmed His criteria which are still used by many pathologists may be tabulated thus (1) the complete destruction of the muscle in and about the base of the ulcer (2) the presence of dense scar tissue not only in and around the base of the ulcer but also within the immediately adjacent subserous coat (3) evidence of obliterative arteritis in the surrounding vessels and (4) fusion of the muscularis mucosae with the fibres of the muscularis propria at the edge of the lesion

The presence or absence of muscle tissue in the floor of the lesion is considered to be of importance in deciding whether a benign ulcer existed before the carcinoma for whereas the muscular coat is destroyed commonly in chronic ulcers it is usual in malignant ulceration for relics of the muscle layer to be dispersed among the infiltrating tumour tissue

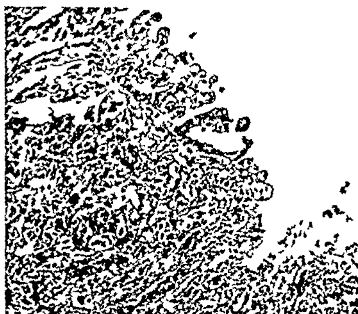


FIG 12
Figure showing superficial origin of carcinomatous tissue at edge of the ulcer H & E × 105

Newcomb (1932) believed that fusion of the muscularis mucosae and the muscularis propria at the margins of an ulcer was a characteristic feature of benign ulcer seldom present in primary carcinomatous ulceration this microscopical feature however cannot be regarded as a reliable criterion since it can be demonstrated in malignant ulcers arising as such *ab initio*

Obviously because of the varying strictness with which these microscopic criteria are applied by different pathologists and because of variations in the nature of the specimens examined and the average age of the patients in the series investigated it is difficult to obtain a true estimate of the frequency with which gastric carcinoma has been preceded by a benign gastric ulcer To prove that an ulcerated cancer of the stomach arose in an antecedent benign lesion is a difficult task no single histological criterion is reliable and even the addition of clinical and radiological evidence may be inadequate to resolve the problem It is however the writer s opinion that it does not exceed ten to fifteen per cent of ulcerated cancers

The examination of established peptic ulcers for evidence of early malignancy (Figs 12 to 15) certainly supports the view that carcinoma only

infrequently develops in the benign lesion The carcinomatous transformation of a peptic ulcer nearly always takes place in its mucosal brim (Fig 12) at separate points which subsequently fuse to form a circumferential ring of malignant tissue The floor of the ulcer rarely is invaded (Fig 13)



FIG 13
The floor of an ulcer-cancer is not invaded by carcinomatous tissue H & E ×65

For this reason the examination of the base of the ulcer to the exclusion of its margins leads to grave diagnostic errors Clearly when the base of a peptic ulcer has been infiltrated extensively it is no longer possible to decide with certainty by histological examination whether a benign lesion preceded the cancer and the probability as already mentioned of such a train of events can be surmised only when there is a large amount of scar tissue in the floor of the ulcerated cancer

Features suggestive of early malignant change in an ulcerative lesion of the stomach include cellular pleomorphism hyperchromatism increased number and atypical mitoses and the formation of solid masses of epithelial

cells and bizarre glandular structures (Figs 14 and 15) Early carcinomatous changes may be difficult to distinguish from the atypical reactive hyperplasia with distortion and displacement of epithelial structures which takes place at or near the edges of a peptic ulcer and although usually the limits of a chronic ulcer are defined sharply now and again a lesion is encountered with irregular and overhanging margins According to Mallory (1940) transitional changes from hyperplastic to normal epithelium begin at the edges of benign

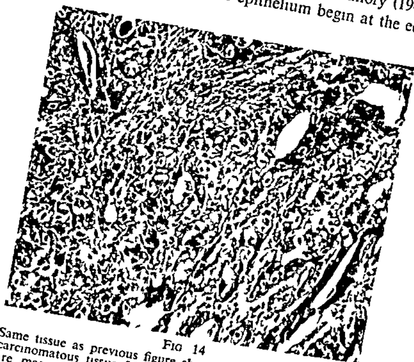


FIG 14
Same tissue as previous figure showing lateral spread of the carcinomatous tissue from the margins of the ulcer There are masses of malignant cells and some bizarre glandular structures H & E × 110

chronic ulcers and these unlike carcinomatous changes are continuous and do not happen in a multicentric fashion Sometimes malignant foci are limited to one small area of a peptic ulcer so that conclusive evidence of their presence or absence may necessitate the examination of serial sections of the whole ulcer Such a procedure is impracticable as a routine measure but several different portions of such a lesion should be examined together with all the available lymph nodes Sometimes the presence of lymph nodal metastatic deposits in the vicinity of an apparently simple peptic ulcer may surprise the pathologist and result in the re-examination of the ulcerative lesion in more detail

The incidence of neoplastic change in a peptic ulcer usually has bee determined by the examination of stomachs resected because of complications evidence of non healing or the situation of the lesion in or near the pyloric

canal Such an expressed incidence is affected by such considerations as the age of the patients (Illingworth 1953) and the number of men and women in such series investigated It is certain that the overall percentage of chronic ulcers in which evidence of malignant change has been demonstrated is small being no more than five per cent and probably less (Morgan and Lee 1954)



FIG 15
Same tissue as previous figure showing focus of malignant tissue in the ulcer margins H & E × 105

Some authorities do not believe that carcinoma ever arises in a benign ulcer (Welch and Burke 1958) although in the past ten to fifteen per cent of ulcerated gastric cancers have been claimed to have arisen on the basis of an antecedent ulcer These figures however demonstrate that at most only a few peptic ulcers become malignant and that the vast majority of ulcerated *gastric cancers do not arise in pre-existing peptic ulcers*

Although a peptic ulcer may also occur in a stomach in which an unrelated cancer exists it might well be that the main influence of a peptic ulcer is to fix the point of maximal tumour potential in a field of gastric mucosa already initiated to undergo neoplastic conversion Such a process would be in accordance with the Deelman phenomenon wherein some traumatic incident *e g* an ulcer determines the site of malignant transformation in an epithelium already in a precancerous state *e g* atrophic gastritis That duodenal ulcers apparently never undergo malignant change is probably due to the nature of the mucosa of the first part of the duodenum which genetically

is hardly ever predisposed to malignant transformation so that an ulcer cannot exercise the same co-carcinogenic effect in this situation as it may do in the stomach

It is perhaps permissible to close this chapter with the observation that in clinical practice primary cancer arising as an ulcer may so improve under conservative treatment as to deceive the clinician into believing the condition to be benign When subsequently the correct diagnosis is made an ulcer-cancer relationship may be assumed erroneously Only the *rapid* and *complete* healing of a gastric ulcer should be accepted as evidence of its benign character failing this resection is advisable Further reference to this clinical aspect of the ulcer-cancer problem is made in Chapters IV and V

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CHAPTER IV

CLINICAL FEATURES

BY IAN W MACPHEE

IN practice the clinical features of gastric and duodenal ulcer are very similar The chief symptom—indeed the only symptom in which the patient is usually interested—is pain There are however some less dramatic symptoms which may be of value in establishing a diagnosis of peptic ulceration The age and sex distribution have already been considered in Chapter I

Pain

As regards the pain they cause both gastric and duodenal ulcers have many features in common The pain frequently commences towards the end of the second or in the third decade of life is dull burning or boring in character and is often related to meals At first the pain is intermittent with long symptom free periods but as the years progress these remissions become progressively shorter Alkalis and in the case of duodenal ulceration food mostly relieve the pain temporarily but once the ulcer has penetrated into an adjacent structure they cease to have any beneficial effect Once penetration has occurred pain sensation is transmitted not only by the splanchnic nerves but also by the parietal nerves the addition of these new pathways with their potentiality for referring pain may alter the type and distribution of the pain experienced by the patient

The pain of a peptic ulcer bears no constant relationship to the degree of activity of the ulcerative process (Cavanagh and Friedman 1956) or to the level of acidity in the stomach and duodenum (Smith 1955) although with a highly acid reaction (pH = 1 8 or less) pain is usually present (Woodward and Shapiro 1954) An ulcer can rarely be completely painless Sometimes the pain is felt in some unusual site and Avery Jones (1957) has stressed the importance of suspecting peptic ulceration as the cause of recurrent pain even when the pain is for instance in the retrosternal area the loins or hypogastrium

GASTRIC ULCER —Gastric ulcer occurs most frequently on the lesser curvature of the stomach The pain variously described as aching boring or even as a lump is ill-defined in the upper epigastrium to one or both sides of the midline It tends to come on ½-¾ hours after meals although its relationship to meals is less constant than with a duodenal ulcer The pain only rarely radiates through to the back unless the ulcer is situated posteriorly and pene

trates the pancreas (Rivers 1947) This pain is not relieved by taking more food though it may sometimes be relieved by alkali especially if this induces eructations Unlike duodenal ulcer the pain of gastric ulcer rarely occurs during the night unless a small hiatus hernia is also present producing the typical retrosternal pain and acid regurgitation on lying down

The periodicity of gastric ulcer pain is not as regular as that of duodenal ulcer although it is customary to suggest that attacks last from 2 6 weeks and are followed by a spell of freedom from symptoms Very large high lesser curve ulcers in old patients are often remarkably painless but if they involve the undersurface of the diaphragm they may cause referred pain in the left side of the neck (Smith 1953)

DUODENAL ULCER —In the early stages of the disease the pain of duodenal ulcer is usually more severe but more easily controlled than that of gastric ulcer *The character of the pain is similar to that of gastric ulcer* and is again ill-defined in the epigastrium and sometimes to the right under the costal margin Radiation to the back is commoner and occurs when the ulcer penetrates the pancreas If the gall bladder or related omentum is involved pain may be felt in the scapular region and with involvement of the hepatic flexure or mesocolon the pain is often referred to the right iliac fossa or hypogastrium (Smith 1953)

The onset of pain is delayed upwards of two hours after a meal and may not occur until just before the subsequent meal So much is this so that patients may not relate the pain to the preceding meal though they are always aware of the fact that the pain can be terminated rapidly by food or alkali The onset of pain is therefore very regular in duodenal ulceration and the patient is frequently awakened during the night

In the early years of the disease the periodicity of pain is fairly regular attacks of pain recurring for a 2 6 week period and being followed by a phase lasting weeks or months during which time no symptoms are experienced Although anxiety and mental stress are so frequently considered as precipi*tating* factors it is interesting to note that the patients in *Barford's (1928)* series considered cold and damp weather as being more important than worry as a cause of onset or recurrence of symptoms Many patients suffer long and unrelieved spells of ulcer pain during severe and inclement weather in autumn and winter Heavy smoking and taking alcohol on an empty stomach usually aggravate the pain of duodenal ulceration but there are some patients who are emphatic that alcohol has no effect on their symptoms With the passage of time the spells of freedom from discomfort in untreated duodenal ulcer become shorter and finally the patient may rarely be free from symptoms even at night when he may get very little sleep

Other symptoms

Direct questioning is often necessary before the patient will mention any symptoms other than his pain However numerous minor symptoms are common With gastric ulceration anorexia and nausea are often present especially with the large ulcers and may rarely be the only symptoms Vomiting can occur too in uncomplicated cases but is more frequent when there is spasm or stenosis causing some degree of obstruction Likewise an uncomfortable full sensation and eructations may be troublesome in a few patients without any obstruction as well as in those with it

Heartburn is a frequent accompaniment of duodenal ulceration Water brash *i e* the sudden filling of the mouth with tasteless or salty clear fluid is of some diagnostic significance being much commoner in duodenal ulceration than in so-called functional dyspepsias (Lees and Rosenthal 1957)

Alterations in the patient s weight or bowel habit are probably not directly due to his ulcer rather are they the result of variations in his diet and medication Vomiting will of course cause both loss of weight and constipation and it is usually found that a patient who is losing sleep is losing weight as well Slight bleeding from an ulcer may cause the stools to become black in colour and if these episodes of bleeding are at all frequent secondary anaemia may develop

Special groups

Peptic ulceration is less common in women than in men and furthermore in most women affected it tends to run a more benign course The symptoms are more readily controlled by medical measures and back and night pain are less troublesome than in men It is not uncommon for an active but symptomless ulcer to be discovered unexpectedly in the course of an examination or operation for some other lesion *e g* gallstones Women often experience a dramatic remission in their symptoms during pregnancy only to suffer a relapse during the puerperium At the menopause there is a slight increase in the incidence and severity of ulceration in women (Clark 1953)

Peptic ulcers can very rarely develop within a day or two of birth and during the first few months of life they mostly present as a free perforation or bleeding (Craver and Glenn 1958) During the next few years of life an ulcer may present with vomiting loss of appetite and failure to thrive but sometimes the child suffers from sudden severe bouts of abdominal pain screams out and vomits (Goldberg 1957) After the age of seven years the clinical picture becomes more like that in the adult but relief by taking food is not so constant Haemorrhage becomes less common but there is often secondary anaemia

In patients over 60 years of age haemorrhage once again becomes frequent and is the presenting symptom in 20 per cent of patients (Cutler 1958) especially in those who have had no previous ulcer trouble Many of these older patients have of course had symptoms for many years and only come to hospital when one of the major complications—haemorrhage perforation or pyloric stenosis—forces them to do so Vomiting and loss of weight are rather commoner than in younger patients and there is a high incidence of associated diseases

Pain is the commonest symptom in patients with giant gastric ulcers but haemorrhage is the presenting symptom in almost one third of such cases (Strange 1959) Ulcers in the pyloric channel do not have any characteristic features to distinguish them from those in the stomach proper or duodenum (Foulk *et al* 1957) while the rare ulcers that are situated beyond the duodenal bulb are often atypical Some may cause pain in the same site as an ordinary duodenal ulcer but the characteristic periodicity and relief by taking alkalis are absent (Rauch 1956) Other post bulbar ulcers first present as bleeding or as intermittent attacks of subacute high intestinal obstruction (Jones 1957 Cooke and Hutton 1958) a few mimic biliary disease but jaundice is very rare

PHYSICAL EXAMINATION

The history obtained from the patient with peptic ulcer is always of greater assistance in the diagnosis than the physical signs to be found Moynihan (1901) in particular stressed that the diagnosis of duodenal ulcer was made entirely on the symptoms described by the patient

It is a common clinical impression that the sufferer from peptic and especially duodenal ulcer is of a lean asthenic build Loss of weight is certainly an occasional feature but Feigenbaum and Howat (1935) have shown statistically that there is no particular bodily type prone to peptic ulcer Ideas to the contrary must be considered as erroneous clinical impressions

Abdominal examination will frequently reveal tenderness over the upper right rectus in duodenal ulcer rarely in gastric ulcer Apart from this there are no characteristic signs of an uncomplicated ulcer Hyperaesthesia and guarding are frequently encountered but persistent gentle examination will usually reveal these to be the result of nervousness on the patient s part In pyloric stenosis the greatly enlarged stomach may be seen to contract and can often be palpated Succussion splash can be elicited but is not of significance within 3 hours of a meal

ANCILLARY METHODS IN DIAGNOSIS

1 Radiological examination

The diagnosis of peptic ulceration is made or at least strongly suspected from the clinical features of the disease Radiological examination by barium meal technique provides confirmatory evidence and is particularly helpful in assisting in the localisation of the site of the ulcer in controlling the progress of medical treatment in excluding malignant lesions in demonstrating the shape of the stomach and duodenum and in estimating at least some aspects of their function Information about the competence of the cardio-oesophageal mechanism can be obtained by appropriate techniques

The radiological features upon which a diagnosis of peptic ulcer may be based are of two kinds visualisation of the ulcer crater and inferential evidence In default of the actual demonstration of the ulcer evidence which suggests its presence has to be accepted The niche of a gastric ulcer can frequently be seen especially if the position of the patient is varied so that oblique views showing the posterior wall of the lesser curvature can be obtained Such a niche is frequently accompanied by a corresponding notch on the greater curvature the result of spasm A benign gastric ulcer crater is usually only 1 2 cm in diameter but can be over 10 cm (Strange 1959) Rarely it may contain a fluid level and small gas bubble of its own (Fig 16) An ulcer niche is less frequently seen in the duodenum and may be difficult to demonstrate especially in a deep-chested patient the commencement of whose duodenum may run more antero-posteriorly than transversely Care must be taken not to mistake a huge duodenal ulcer crater seen *en face* for a normal duodenal cap

The indirect or inferential evidence of the presence of an ulcer consists of the demonstration of distorted mucosal pattern tenderness spasm abnormal peristalsis and deformity In assessing the value of these pieces of evidence the screening of the patient is much more important than the spot films When the first mouthful of barium suspension has been swallowed the mucosal pattern can be outlined With a simple ulcer the converging mucosal folds extend right up to the edge of the ulcer (Kirsh 1955) Tenderness at the point of convergence of the folds or tenderness over a deformed duodenal cap is almost diagnostic of ulceration even though a crater cannot be demonstrated With a duodenal ulcer the gastric mucosal folds are often prominent Spasm of the wall (usually the greater curvature) opposite the presumed site of an ulcer gives further confirmation of its presence Sometimes spasm of the pylorus causes an increase in the volume of resting juice and delays the exit of barium from the stomach This is considered further

in Chapter X but here it may be stated that pylorospasm can be caused by an ulcer in the stomach in the pyloric canal or commonly in the duodenum Abnormally frequent and deep peristaltic waves are often seen sweeping down



FIG 16
Giant ulcer on the lesser curvature of the stomach Note the air bubble in the ulcer crater and the very marked indrawing of the greater curvature (*By courtesy of Dr P Whitaker*)

the stomach especially when there is some element of obstruction present With stenosis there is mostly considerable dilation of the stomach and a large 6 hour gastric residue

Deformity due to cicatricial contracture and not to spasm is the result of long standing ulceration In the stomach if the contraction is mainly transverse it causes the now rare hour glass deformity in which the greater

curvature is gradually indented and drawn over towards the lesser curvature if the contraction is longitudinal the lesser curvature may be so shortened and drawn up that the duodenum appears to arise from halfway down it Distortion and irregularity of the duodenal cap may be due either to spasm in which case it diminishes with medical treatment, or to scarring when it persists unchanged often having a trefoil shape Both features are characteristic of duodenal ulceration Deformity of the duodenal cap can however be caused by previous inflammation in or operations on adjacent structures such as the gall bladder With a duodenal ulcer it is not uncommon to find a pseudodiverticulum present between the pylorus and the ulcer

Ulcers in certain situations are notoriously difficult to demonstrate Because of gravity the fundus and the roof of the pyloric antrum often do not fill properly with the patient in the erect position The head-down position must be utilised to resolve any doubts about a lesion in these areas and this position also serves to demonstrate any hiatal hernia present and the competence or otherwise of the cardia The rapid transit of barium through the second part of the duodenum mostly makes it difficult to show up the rare ulcer in that site Likewise in children it may be very difficult properly to outline any part of the duodenum such is the rate of passage of the barium suspension through it In both these groups and in any other doubtful cases repeat examinations after some weeks may be necessary However when considering the advisability of further X rays the dangers of excessive irradiation must be balanced against the severity of the patient s symptoms It is not justifiable to have frequent barium meal examinations for minimal or dubious symptoms

The radiological investigation of an ulcer patient is completed by having a straight X ray of his chest to exclude any active or quiescent pulmonary disease or any lesion which might impede the passage of a gastroscope

Tests of gastric secretion

There is no really satisfactory test of gastric secretion which is suitable for routine hospital use The old gruel fractional test meal is useless and nauseating and may be actively misleading Small doses of alcohol caffeine and histamine (0 5 mg histamine acid phosphate) have been given as the gastric stimulant instead of gruel but do little to increase the accuracy of the test The tip of the Ryle s tube may be badly positioned and may not be in the stomach at all—this is a criticism that applies to all the tests in which gastric aspiration is used unless X ray control is employed in positioning the tube The tip of the tube should be radio-opaque The aspirations must be performed frequently every 10 or 15 minutes otherwise a significant propor

tion of the gastric secretion passes onward from the stomach without being aspirated The best yield or recovery rate of gastric juice is obtained by using continuous *hand* suction with the patient sitting up (Johnston and McCraw 1958) and this is to be recommended for relatively short periods of aspiration For longer periods the modification of the apparatus originally described by Sequeira (1954) and shown in Fig 17 is admirable A small readily available home aquarium pump provides suction and the 6 inch water seal in the valve bottle prevents excessive negative pressure being built up

During the past decade two tests of gastric secretion have been developed which avoid the necessity of passing an aspiration tube but unfortunately neither of them can be recommended in the routine investigation of patients

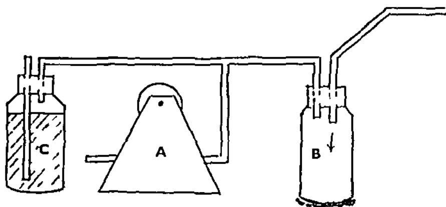


Fig 17
Gastric suction apparatus using an aquarium pump (A) The gastric contents are drawn into bottle (B) while the level of the fluid in the valve bottle (C) controls the negative pressure produced

with peptic ulceration The first of these is the *uropepsin test* (Sircus 1954) This is a measure of the secretion of pepsin in the stomach and is not necessarily a reliable index of acid secretion While the urinary output of uropepsin is generally considerably increased in duodenal ulceration wide variations may be found in individual patients (Kyle 1956 Myerson and Corazza 1957) and the slightest variation in the complicated technique or in the reagents used in the uropepsin estimation alters the result markedly The second test is the so-called *tubeless gastric analysis* (Segal *et al* 1950) A test compound (marketed as Diagnex by Squibb) which consists of an ion exchange resin combined with either quininium or azure A dye is used Two grammes of Diagnex are swallowed after an overnight fast and 0 5 mg histamine are given In the stomach a small percentage of the quininium or azure A is set free by the hydrochloric acid and is then absorbed and excreted in the urine which is collected for 2 hours This Diagnex test is purely *qualitative* for the presence of free acid and has never been claimed as a

quantitative estimate of acid secretion (Segal *et al* 1953 Conway and Meikle 1953 Denborough *et al* 1958) After gastric operations the Diagnex granules may not be in a sufficiently acid medium for long enough (the pH must be under 3) for the quininium or dye to be released (Conway and Meikle 1953 Shay *et al* 1954) Pyloric stenosis or diarrhoea make the Diagnex tests useless and their results may also be upset by cation-displacing ions such as barium magnesium calcium and iron In the more complicated quininium variant the results may be invalidated by such drugs as quinine and the vitamin B complex Intestinal malabsorption and liver and renal disease interfere significantly and unpredictably with the excretion of quininium or azure A in the urine (Behr and Lawrie 1955 Fentress and Sandweiss 1957 Bolt *et al* 1957)

Three special tests of gastric function are of value in the investigation of patients with peptic ulceration They are the augmented histamine test of Kay (1953) the nocturnal secretion and the insulin test meal

1 *Augmented Histamine Test*—after a 12 hour fast the patient swallows a radio opaque Levin tube size 12 or 14 Charriere The tip of the tube is placed under X ray control in the most dependent part of the stomach the patient lying comfortably on his left side The fasting juice is aspirated and discarded and the basal juice then collected for 30 or 60 minutes by continuous suction At the end of this period 4 ml of mepyramine (Anthisan May and Baker) are given intramuscularly and half an hour later histamine acid phosphate 0 04 mg /kg body weight is injected subcutaneously Fifteen minutes later any juice that has collected since the end of the basal secretion period is removed and the maximally stimulated secretion is collected for 30 or 60 minutes The free acid and chloride contents of the basal and of the stimulated secretion are then estimated

Kay (1953) found that the average maximum output of HCl during a 30 min period was 12 mEq in normal subjects 13 mEq in gastric ulcer patients and 23 mEq in duodenal ulcer patients With very high outputs of acid *e g* over 28 mEq radical surgery is needed if recurrent ulceration is to be avoided

2 *Nocturnal Secretion*—the continuous aspiration of juice for a 12 hour period through the night is a relatively simple test to perform but the position and patency of the tube cannot be assured during so long a period unless there is close supervision In normal subjects and in patients with single uncomplicated gastric ulcers the all night secretion of free HCl is below 10 mEq in 80-90 per cent of cases (Johnson 1955) while in 60-70 per cent of patients with duodenal or combined duodenal and gastric ulcers it is over 10 mEq of

HCl In duodenal ulcer patients the volume of night secretion is usually just over one litre which is more than double the output of normal people (Levin *et al* 1950)

3 *Insulin test meal*—this is a test primarily for the integrity of the vagus nerve pathway from the hypothalamus to the stomach It is however influenced by the responsiveness of the posterior hypothalamic centres to a fall in the blood sugar level and recently it has been suggested that part of the response to this fall is mediated by the pituitary adrenal axis (Stempien *et al* 1958) Fifteen units of soluble insulin are given slowly intravenously and 15 minutes later after emptying the stomach the gastric secretion is aspirated continuously for one hour The insulin must produce a reasonably rapid fall in the blood sugar level down to about 50 mg per cent A specimen of blood should be taken just before and at the end of the test period for estimation of the blood sugar level and concentrated glucose solution must be available for injection if the hypoglycaemia symptoms are unduly severe

Unfortunately in practice the result of the insulin test is often vitiated by the presence of an artificial stoma in the stomach Such surgical openings can only be closed by elaborate balloon blocking techniques not suitable for routine use Little importance can therefore be attached to negative results but the presence of any quantity of free acid (over 1 mEq /hr) usually indicates that vagotomy has been incomplete

Occult blood

Any breach in the mucosal lining of any part of the alimentary canal causes occult blood to appear in the stools but the blood loss is intermittent and 5 20 ml of blood must have been shed into the lumen of the gut before most of the routine tests on the faeces will detect it In peptic ulceration occult blood tests are positive in up to 80 per cent of patients (Barford 1928) but the test often has to be repeated several times before a positive result is obtained No importance can be attached to a negative result no occult blood may be detected in the faeces when there is an active ulcer (Jones 1957) Obvious sources of blood loss such as bleeding gums or haemorrhoids must be excluded before carrying out the test

The original benzidine test (Adler and Adler 1904) is little used nowadays With concentrated (8 per cent) solutions of benzidine it frequently detects minute traces of blood in the faeces of normal people it is necessary to avoid meat and especially liver for 3-4 days before the test (Thornton and Illingworth 1956) and there are some carcinogenic risks attached to the manufacture of benzidine (Smith 1958) The slide technique for performing the benzidine test (Gregersen 1919 Harrison 1957) is simple clean and does

not normally require any dietary restrictions beforehand (Needham and Simpson 1952) When a quick positive reaction is obtained (deep blue colour developing in 15 secs) there are very few false positive results If the result is equivocal it can be repeated after a few days on a meat free diet

To avoid the use of benzidine in these tests orthotolidine has been introduced as the reagent (Kohn and O Kelly 1955 Smith 1958) With a 0 2 per cent solution of orthotolidine very few false positive results are obtained and the only dietary preparation necessary is to avoid foods such as liver which contain sizeable amounts of blood A tablet variant of this test is now available using Occultest or Haematest tablets (Ames Co (London) Ltd) The Occultest tablets are the better ones for routine ward use the colour change being easier to read than that of the Haematest tablets (Smith 1958) Weakly positive tests should either be ignored or repeated

The actual amount of blood (if over 3 ml) being lost into the gut lumen can now be measured by isotope techniques using radioactive chromium (Bannerman 1957) Although requiring special apparatus and precaution this test serves as a useful check on the accuracy of other simpler techniques for detecting occult blood in the faeces

Gastroscopy

Gastroscopy is an ancillary procedure only occasionally required in the diagnosis of peptic ulceration The principal indications for its performance are given by Rodgers (1947) as follows

1 When clinical and radiological examinations have failed to reveal the cause of dyspeptic symptoms

2 To help differentiate between a benign and a malignant ulcer of the stomach

3 To follow the healing of a gastric ulcer during medical treatment (Fig 18)

4 In the investigation of recurrent symptoms following operations on the stomach

It should be noted that gastroscopy is not of direct help in the diagnosis of a duodenal ulcer except to exclude the presence of a concomitant gastric lesion Due to scarring and posterior penetration a duodenal ulcer frequently so distorts the antrum and dislocates it posteriorly that it is not possible to visualise its interior with the gastroscope

In Britain there are two types of gastroscope in common use the smaller Wolf Schindler (maximum diameter 12 mm) and the larger Schranz Taylor (maximum diameter 14 mm) instrument the tip of which can be flexed through 30 by the operator To pass such a semi rigid instrument safely

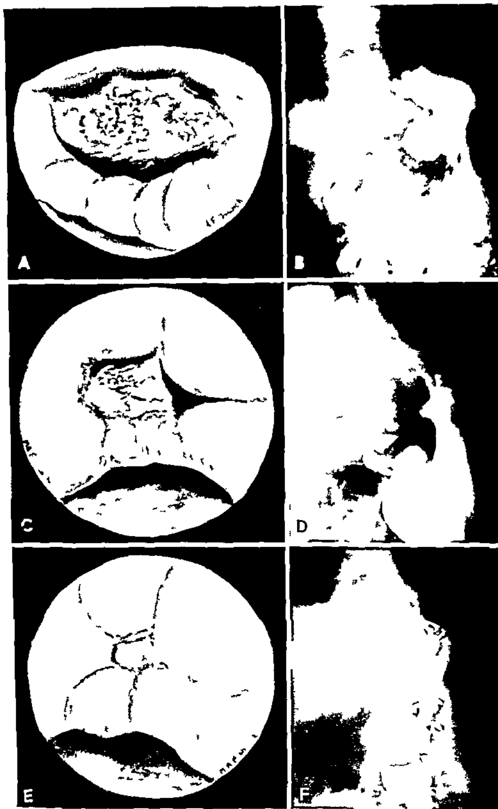


FIG 18

Observing the healing of a gastric ulcer by repeated gastroscopic and radiological examinations (*By courtesy of Professor H W Rodgers*)

(A and B) Giant lesser curvature gastric ulcer before treatment

(C and D) Appearances of the same ulcer after four weeks of medical treatment The ulcer is only one third of its original size

(E and F) Continued shrinkage of the ulcer crater as observed after a further four week period of treatment

through the delicate pharynx and oesophagus requires considerable skill and experience It should never be attempted when any active pathology is present in the oesophagus Before instrumentation the physical examination and study of the chest X ray and barium meal films should have excluded *any lesion such as an aneurysm or cervical osteophytes* causing extrinsic pressure on the oesophagus or any fixed kyphosis or abnormality of the teeth and jaws which would render the *investigation dangerous or impossible* In women over the age of 50 the upper alimentary mucosa is frequently more delicate and atrophic than in younger women or men and in older women it is therefore advisable to use the smaller Wolf Schindler instrument

Most gastroscopies can be performed with sedation and local anaesthesia and Taylor (*1958*) often relies on premedication alone Atropine *1/100* gr and morphine ¼ gr are given 90 minutes before the examination or alternatively 100 mg pethidine can be *given intravenously on the operating table* (Rodgers 1956) The premedication is supplemented by spraying the patient s lips gums and posterior pharyngeal wall slowly with 2 ml of 1 per cent amethocaine hydrochloride and getting him to work this round his mouth and then swallow it General anaesthesia (Goligher and Thornton 1951) is usually only needed for very nervous patients

The patient lies on his left side on the operating table with his knees *drawn up to a right angle (Rodgers 1956) His head is supported by a* strong and intelligent assistant who keeps the head slightly extended without drawing it backwards The operator holds the distal end of the lubricated gastroscope like a pen the proximal end can either be supported on his shoulder or by a nurse With the index and middle fingers of his left hand he gently hooks the patient s tongue forwards and *slides the tip of the instrument* over the back of the tongue between his two fingers When the tip reaches the posterior pharyngeal wall it can be bent caudally and slightly forwards by the index finger and the patient is then asked to swallow once or twice This should enable the gastroscope to enter the oesophagus smoothly and without resistance If it does not go in readily the attempt should be abandoned force must never be used for fear of damaging the posterior pharyngeal wall *Careful watch must also be kept for any resistance* when the tip of the gastroscope reaches the lower end of oesophagus and only the gentlest rotatory movements used to coax the tip through the cardia which however it usually passes through without difficulty

The examination of the interior of the stomach is conducted in a systematic manner taking the pool of mucus on *the dependent greater curvature* as a reference point The instrument is advanced slowly with slight air distension of the stomach and both curvatures and walls are closely inspected In the lower third of the stomach particular note is made of the activity of

the antrum and of the pyloric sphincter which is not always visible More air is then pumped into the stomach and the various areas inspected as the instrument is gradually withdrawn It may be difficult or impossible to get a good view of the fundus of the stomach of the area immediately beyond the tip of the instrument of the antrum (and especially of its roof in a J shaped stomach) and sometimes of the uppermost part of the lesser curvature which may be too close to the objective lens These difficult areas can often be brought into view by flexing the tip of the Schranz Taylor gastroscope by varying the degree of air insufflation and by the operator using his left hand to press gently on the patient s abdominal wall and indent various parts of the stomach A complete view of the interior of the stomach and also of the duodenum is said to be obtained with the new fiberscope a gastroscope in which the image is transmitted not by a series of lenses but by an elongated bundle of fine glass fibres (Hirschowitz *et al* 1958)

At the conclusion of the examination when the instrument is withdrawn it is carefully wiped to see if there is any blood on it which would suggest damage to the pharynx or oesophagus The patient is not allowed to eat or drink until the local anaesthesia in the pharynx has worn off

Serious complications after gastroscopy are uncommon developing in from 0 1 to 0 5 per cent of cases (Jones *et al* 1951 Smith and Tanner 1956) The commonest injury is damage to the posterior wall of the hypopharynx and upper end of the oesophagus probably from pressure necrosis Damage can follow what had appeared to be an easy and uneventful examination (Taylor 1958) and is commoner in women The fascial investment of the pharynx and oesophagus is usually intact at first and only sloughs after some hours Peri-oesophagitis then develops causing local pain and tenderness dysphagia pyrexia and a stiff neck Treatment is at first conservative with parenteral feeding and antibiotics Only if suppuration occurs is it necessary to drain through an incision along the lower part of the posterior border of sternomastoid Frank perforation of the lower oesophagus or stomach is even rarer than damage from pressure of the instrument against the cervical spine That perforation has taken place may be obvious on looking down the gastroscope or by failing to see anything or subsequently finding that the instrument is heavily blood stained but rarely it may occur unexpectedly many hours after the gastroscopy Treatment is by prompt thoracotomy or laparotomy and repair of the perforation

DIFFERENTIAL DIAGNOSIS

By careful consideration of the clinical history and the results of the special investigations especially the barium meal it is possible to make a correct diagnosis of a gastric or of a duodenal ulcer in the vast majority of

cases Due attention should be paid to the site and type of the pain to its radiation and to the factors which aggravate and which relieve it It is well to remain suspicious of peptic ulceration in any patient who complains of recurrent abdominal or lower chest pain even when the pain is in an unusual site (Avery Jones 1957) Repeated radiological examinations may be neces sary to establish the diagnosis

Clinically it is frequently impossible to differentiate between a gastric and a duodenal ulcer but some of the points which occasionally help are mentioned on p 56 Accurate localisation of the ulcer frequently has to await the radiological demonstration of its crater Both types of ulceration may co-exist The differentiation between a benign and a malignant gastric ulcer is considered in detail on p 72 Some of the other conditions which may have to be distinguished from peptic ulceration are considered briefly below Again it should be remembered that ulceration is commoner in patients with many of these conditions than it is in the general population

1 *Diaphragmatic hernia*—Of the three types of diaphragmatic hernia the type which slides through the oesophageal hiatus is the one most likely to be confused and also to co exist with peptic ulceration usually in the duodenum A retrosternal burning sensation worse on stooping or lying flat acid regurgitation flatulence and sometimes dysphagia should suggest the correct diagnosis The uncommon paraoesophageal hernia may cause cardiac symptoms and the patient may with reason complain of wind around the heart Frequently there is a gastric ulcer in or at the edge of the herniated pouch of stomach it can bleed alarmingly The rare congenital herniations through the foramina of Morgagni and Bochdalek are only likely to be diagnosed on barium meal examination or at laparotomy

2 *Functional dyspepsias*—This group includes numerous ill understood inflammatory and atrophic changes involving the gastric mucosa Some are chronic or recurrent others appear to have an allergic basis while a few follow known indiscretions such as excessive ingestion of alcohol or aspirin Nausea vomiting and anorexia are much commoner than in simple ulceration the patient usually feels epigastric discomfort or distension rather than pain and flatulence is common If pain is felt it may be made worse by food Bleeding is quite common A correct diagnosis can often only be arrived at after full investigation with X rays gastroscopy histamine test meal and even gastric biopsy (Williams *et al* 1957)

3 *Cholecystitis*—Typical flatulent dyspepsia in an obese middle aged female is unlikely to be confused with ulcer disease but chronic cholecystitis in men or thin women can be more difficult to diagnose correctly The pain

of gall bladder disease has usually at times a colicky character and is situated more to the right of the upper abdomen radiating to the right scapular region—an unusual distribution for ulcer pain A cholecystogram and an intravenous cholangiogram (40 ml) may be necessary and it should be remembered that the inflammatory reaction around a diseased gall bladder may cause considerable distortion of the duodenal cap as seen on a barium meal

4 *Pancreatitis*—Acute pancreatitis may simulate perforation of an ulcer (see p 148) but may be differentiated from it by the less marked muscular rigidity greater degree of shock by marked elevation of the serum amylase level and by the absence of gas under the diaphragm Chronic pancreatitis is a diagnosis which is seldom made because it is seldom thought of The pain tends to be severe and chronic with occasional exacerbations and is felt mostly in the back Often the patient sits up and bends forward in an attempt to gain relief from the pain Food and alkalis are ineffective in bringing relief Weight loss can be marked and examination of the faeces reveals evidence of impaired fat and protein digestion

5 *Cirrhosis*—Chronic indigestion rather than pain is the main alimentary feature of cirrhosis Food may make the discomfort worse and there is often other evidence of the liver disease such as ascites tiny spider naevi on the face a sweet breath and erythema of the eminences of the palms Ulcers may develop in the stomach or duodenum of cirrhotic patients while bleeding from oesophageal varices may simulate that from an ulcer

6 *Duodenal diverticulum*—A congenital diverticulum may cause vague indigestion and flatulence it can cause acute symptoms by bleeding or by becoming inflamed or causing obstruction It is usually only detected on careful radiological investigation if in addition an ulcer crater is seen the patient s symptoms are usually due to the ulcer and not to the diverticulum which should only be removed if there is clear objective evidence that it is causing trouble

7 *Appendicitis*—The diagnosis of chronic appendicitis is less popular now than formerly True recurrent abdominal pain especially if felt in the upper abdomen and relieved by food is much more likely to be caused by a peptic ulcer Appendicular pain is usually of colicky type and felt at the umbilicus or in the right iliac fossa Rarely however ulcer pain may be felt low down in the abdomen

8 *Carcinoma Coli*—Occasionally in a pale elderly patient a barium meal does not show a gastric ulcer that had been suspected clinically In

these patients it is important to exclude a colonic lesion by abdominal and barium enema examinations and by sigmoidoscopy The patient should be carefully questioned about any alteration in bowel habit or rectal bleeding Occasionally the distension discomfort and anorexia of simple chronic constipation may simulate gastric ulceration in a younger patient

9 *Tabes*—The gastric crises of tabes though rare closely mimic an acute exacerbation of duodenal ulcer and even perforation The acute pain tenderness and rigidity in the epigastrium may be the same as in ulcer patients A careful neurological examination may reveal that the pupils are small irregular and do not react to light that the ankle and knee jerks are absent that stereognostic sensibility is deficient in the lower limb and that pain sensibility is diminished or lost Trophic changes in the joints of the lower limb may be found Often some of these signs are absent and great care is necessary especially since peptic ulceration is common in tabetics The possibility of a true perforation in a tabetic patient (or in a patient after thoraco-lumbar sympathectomy) must not be overlooked

10 *Chest disease*—Pulmonary lesions may occasionally present features comparable in some respects with those of peptic ulcer Chest pain from any disease of the lungs or pleura may radiate across the epigastrium This pain however is not related to meals and differs in character from that of peptic ulcer being frequently worse on coughing or hyperventilation Pulmonary tuberculosis too may be associated with true indigestion intestinal upset vomiting and weight loss If suspected a radiogram of the chest should provide the diagnosis but if unsuspected an extensive investigation of the stomach and duodenum may be carried out before ulcer is excluded

11 *Heart disease*—In cardiac failure epigastric discomfort after food may occur but the discomfort usually arises as a result of the meal and is not relieved by it Retrosternal pain arising from this cause is made worse by eating or after effort and in these respects is quite unlike ulcer pain Dyspnoea and congested neck veins are obvious warning signs

The Distinction between Benign and Malignant Ulceration of the Stomach

The history of duodenal ulceration is so typical that confusion with neoplastic disease of the stomach scarcely occurs The differentiation between chronic gastric ulcer and some forms of gastric carcinoma may however be impossible even at laparotomy The reasons for this are obvious In both conditions the history may be short the symptoms vague a large ulcer may be present associated with gastritis and achlorhydria and loss of weight and decline in health may be evident It is true that many large peptic ulcers show

evidence of healing with treatment whereas the progress of gastric carcinoma is usually relentless Such evidence can however only be obtained by fairly prolonged treatment and observation and may be misleading since careful nursing may relieve all signs and symptoms temporarily even in carcinoma If the features of gastric carcinoma finally become evident valuable time will have been lost and a life may have been jeopardised for purely academic considerations Procrastination is therefore totally unjustifiable The annual reported deaths from gastric carcinoma yearly in England and Wales amount to almost 15 000 (Registrar General 1951) If this excessively high death rate is to be reduced by surgery early diagnosis and operation are necessary By these means diagnostic mistakes will certainly occur and subtotal gastrectomy will be carried out on a number of patients with a chronic peptic ulcer which would probably have healed with medical measures The price paid by the patient is not however too high The end results of subtotal gastrectomy for peptic ulceration of the stomach are good The patient is assured a permanent cure and will be spared the possible complications of ulceration namely haemorrhage and perforation Radical treatment is therefore in the patient s best interests The greatest difficulty and the one requiring most judgment arises when the lesion is situated at the cardia Under these circumstances radical treatment for cancer implies cardio-oesophagectomy at least This treatment carried out for a simple peptic lesion would occasion grave misgiving as the disability associated with proximal or total gastrectomy is considerable and is mostly unjustified in peptic ulceration These lesions therefore require especially judicious consideration and temporisation for a brief period may occasionally be justifiable With this proviso where serious doubt persists after investigation the treatment of a chronic gastric ulcer is early operative resection with subsequent histological examination of the ulcer and related lymph nodes

CLINICAL DIFFERENTIATION —The age and sex of the patient is of little assistance in differentiating between a benign and a malignant lesion Both are commoner in men and nearly a quarter of all gastric carcinomas occur in patients under fifty years of age (Swynnerton and Truelove 1951) The history too is of little help In both conditions the patient may never previously have suffered from indigestion In both conditions the onset may be insidious and even if digestive symptoms are present they are similar Vague epigastric discomfort experienced chiefly after meals and associated with flatulence nausea or vomiting are common The loss of appetite may be more distinct and more specific for protein meals in carcinoma than in peptic ulcer Perforation haematemesis melaena and occult blood in the stools occur in each condition The loss of weight and the deterioration in health are more marked

in frank neoplastic disease but the weight changes in benign and malignant ulcers are very similar (Smith *et al* 1953) *in chronic peptic ulcer weight loss* may be considerable when pyloric stenosis has occurred The signs of dissemination of carcinoma—palpable metastatic deposits in the abdomen pelvis or neck glands enlargement and irregularity of the liver ascites and jaundice—these all indicate that the end is in sight and are not considered *here*

Radiological and gastroscopic investigation are the most valuable aids in diagnosis

1 Radiological features —A massive filling defect or linitis plastica are evident features of carcinoma *The chief problem is to differentiate a* malignant from a simple ulcer Assistance may be gained from a consideration of the following features

(a) Site —Both types of ulceration occur commonly in the antrum on the lesser curvature and high in the fundus Ulceration occurring on the greater curvature however is said to be malignant in only about fifteen per cent of cases (Marshall 1953, Bernardo *et al* 1958) Of ulcers in the pyloric region of the stomach between five to twenty per cent are malignant (Marshall 1953 Smith *et al* 1953 Welch and Burke 1958) Although the incidence of malignant ulcers in both these sites is much lower than formerly believed it is still sufficiently high to justify resection

(b) Outline —A large elevated ulcer with deeply rolled edges must be considered carcinomatous Any filling defect in the stomach even with a clear ulcer niche or a deformity due to para gastric glands is always very suggestive of malignancy as are irregular distorted mucosal folds not reaching the edge of the ulcer (Kirch 1955)

(c) Size —The great majority of giant (over 4 cm diameter) gastric ulcers are benign (Jennings and Richardson 1954 Cohn and Sartin 1958) *Malignant ulcers show a greater variation in size than benign ones* (Marshall 1953) and can be very large but as they only form ten to fifteen per cent of all ulcers in the stomach most of the large ulcers seen on X ray should be regarded as probably benign

(d) Gastric motility —The value of screening in the examination of gastric peristalsis is considerable The interruption in the wave of contraction around a gastric lesion is often indicative of carcinoma Even the appearance of rigidity and fixity of the lesser curvature of the stomach must be considered with grave suspicion However many large simple peptic ulcers at this site may be surrounded by a wide zone of dense oedematous and inflammatory

chance and may have penetrated the pancreas Such lesions also produce rigidity and fixity of the stomach wall so that none of the features is diagnostic

(e) *Multiplicity* —Multiple benign gastric ulcers are not uncommon but it is rare to find any other ulcer in a stomach which contains a malignant lesion (Dolphin *et al* 1953) It is also very uncommon for there to be a duodenal ulcer present at the same time as a malignant gastric ulcer

(f) *Calcification* —There is an unusually low incidence of calcification in the major abdominal arteries in gastric carcinoma such calcification is common with benign ulcer (Elkeles 1956 Dungal and Benediktsson 1958) To what extent this radiological sign may be of value in differentiating benign from malignant ulcers (as distinct from obvious gastric carcinomata) is still undetermined

(g) *Healing* —If the physician and surgeon decide to try a short therapeutic test with medical treatment several radiological examinations during the course of four to six weeks should show progressive healing of the ulcer and finally its complete disappearance Many malignant ulcers will show some diminution in size but they rarely disappear completely A check barium meal three to four months after apparent healing is always advisable to make sure there has been no recurrence of ulceration—always a suspicious feature

The radiologist s task in differentiating a benign from a malignant ulcer is often difficult and his responsibility is grave Estimates of the percentage of correct radiological interpretations vary from fifty four per cent (Dagradi and Johnson 1957) to ninety per cent (Jordan 1958 Bernardo *et al* 1958)

2 Gastroscopic examination —To distinguish between a benign and a malignant ulcerated lesion in the stomach by gastroscopy requires considerable experience Features suggestive of malignancy are a raised irregular edge to the ulcer an uneven necrotic and often blood stained floor especially after medical treatment and rigid and deformed folds in and an elevation of the whole ulcer above the surrounding mucosa (Rodgers 1948) Gastroscopy is not necessary in every case of gastric ulcer it is used to provide additional information when after adequate radiological examination there is still doubt about the nature of the lesion It is accurate in from fifty to seventy per cent of cases and when the gastroscopic and radiological findings are considered together a correct diagnosis can be made in over ninety per cent of cases

3 Examination of gastric contents —The retention of large quantities of foul smelling fluid in the stomach and the presence of blood and

sarcinae are *not* reliable evidence of gastric malignancy The absence of free hydrochloric acid on a gruel test meal is also unreliable and at best indicates gastritis which may be associated with either a benign or a malignant ulcer With histamine stimulation true achlorhydria is rare if it is found it indicates that there is an atrophic type of mucosa which is the type most likely to undergo malignant change Many malignant ulcers however are found in stomachs secreting free acid although the output of acid and chloride on maximum stimulation with histamine is greater with benign ulcers than with gastric carcinomata An H ion concentration above 80 mEq /L combined with a Cl concentration above 135 mEq /L is said to be diagnostic of benign ulceration in 100 per cent of cases (Hirschowitz and Wiggins, 1957)

The examination of gastric washings by the Papanicolau technique is seldom helpful in differentiating between a benign and a malignant ulcer (Burnett 1959 personal communication) Many false negative results are obtained but the demonstration of typical carcinoma cells is positive evidence of gastric malignancy (Hennig and Harvey 1959)

4 BLOOD CHANGES —Secondary anaemia as a result of small repeated haemorrhages occurs with both lesions The normochromic or hypochromic anaemia tends however to be more severe and more resistant to treatment when a carcinoma is present The association of an ulcer in the stomach and a megaloblastic Addisonian anaemia is very suggestive of malignancy and the diagnosis should be made by the pathologist on the resected specimen

Conclusion

It is often possible to tell radiologically whether an ulcer in the stomach is benign or malignant If there are features suggestive but not diagnostic of malignancy the ulcer should be resected and surgery should also be used for a recurrent ulcer or for one in an unusual site When there is no radiological evidence that the ulcer is malignant but the history or other clinical findings suggest that it may be the patient should be gastroscoped and the gastric response to maximal stimulation with histamine determined If doubt still exists about the nature of the lesion two courses of action are available (1) a gastric resection is carried out in accordance with the operative findings *or* (2) the progress of the patient and his ulcer under medical treatment can be watched radiologically and gastroscopically for one month If the ulcer is benign healing should be rapid complete and permanent However even should temporary improvement appear to take place carcinoma cannot always be excluded and it is therefore necessary to be very strict in the

application of this therapeutic test The criteria of healing of an ulcer are improvement in the patient's clinical state cessation of bleeding and above all the shrinkage of the ulcer crater and the return of its outline towards normal on radiological and gastroscopic examination If unequivocal evidence of healing is lacking at the end of a month the lesion *must* be explored without further delay

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CHAPTER V

THE PRINCIPLES OF SURGICAL TREATMENT OF PEPTIC ULCER

By CHARLES WELLS

PEPTIC ulceration is a very common complaint but not all patients suffering therefrom require operation Avery Jones (1957) has estimated that about one half of the patients with gastric ulcer and one third of those with duodenal ulcer need surgical relief in the more northern parts of the country an even larger proportion of duodenal ulcer patients may eventually come to operation

For present purposes it may be assumed that non-operative treatment belongs to the realm of the physician and the details of such treatment are beyond the scope of this work In general however it may be said that intensive medical therapy involves complete bed rest and the cessation of smoking Complete mental and physical rest if they can be achieved often lead to prolonged relief of symptoms The value of special diets and medicaments is very doubtful but during acute exacerbations of ulceration a light diet or milk drip alkalis and belladonna are all useful in relieving the acute pain and discomfort The long term medical management of peptic ulceration includes continued abstention from smoking and often entails the abandoning of a hard working competitive career in favour of a life lived at a slower pace and in more peaceful surroundings Such a regime is unfortunately all too often either not possible or not acceptable The greatest drawback to medical therapy however is the fact that three-quarters of the patients eventually suffer from recurrence of ulceration with the possibility of serious complications developing

INDICATIONS FOR OPERATION

When discussing the indications for surgery it is necessary to anticipate the end of the story because the readiness with which an operation is advised depends upon the risks and the prospects of success which attend it In every patient the chances of success or failure of operation must be balanced against the degree of suffering and disturbance of normal life which his ulcer is causing him It must therefore be stated at once that the prospects of operative treatment already good are continually improving The operative mortality in a well-organised centre is between one and two per cent which is certainly much less than the risk of a fatal complication developing in untreated ulcera

tion A worthwhile indeed a good or a well nigh perfect result is achieved in almost ninety per cent of patients submitted to surgery and these patients require no further treatment The time and attention rightly devoted to the small percentage whose results are seriously disappointing must not be allowed to obscure the benefits available to the great majority

Bearing these premises in mind the indications for operation can now be considered Frequently the clearest indications are those present during the acute complications of perforation haemorrhage and pyloric stenosis These are discussed fully in Chapters VIII IX and X They will therefore not be dealt with here except to state that the past history of one of these complications is a powerful argument in favour of operation and a history of repeated complications is an absolute indication for surgery

SEVERITY AND DURATION OF SYMPTOMS —In uncomplicated peptic and especially duodenal ulceration before deciding in favour of operation a variety of factors require careful consideration The first of these is the severity of the symptoms There is no doubt that post gastrectomy troubles are more likely if a patient with only mild symptoms is submitted to operation (Illingworth 1953 Capper and Welbourn 1955 Pulvertaft 1958 Mackenzie *et al* 1958) Such a patient has never known what real suffering is and in consequence may complain excessively about the discomforts which sometimes follow gastrectomy Ogilvie (1952) and Kinsella (1956) have stated that the poor results of gastrectomy are due to faulty technique but in some cases they are due to faulty selection of patients It is sometimes said that the patient should be made to earn his gastrectomy (Thomson 1957) and that surgery should not be contemplated unless severe persistent symptoms have been present for many years This argument however can be pressed too far and one of the principal duties of any doctor is to alleviate suffering not to see how much his patient can tolerate *Once a reliable type of patient has in spite of medical measures begun to lose sleep on account of night pain or has become unfit to do his work properly then the time has come to advise operation*

Closely related to the severity of the symptoms is their duration An ulcer which has only been present for a few weeks or months may well be of the acute superficial type which will respond rapidly to medical measures Furthermore in the early stages of ulcer disease there are generally long remissions without any pain In these circumstances it would be folly to perform gastrectomy and remove the major portion of an organ all of whose functions are as yet not known Once a duodenal ulcer has been present for two to three years the remissions tend to be shorter and less frequent and the chances of medical treatment ever achieving a permanent cure are very

much less Surgery is therefore more likely to be necessary From some patients a long history of ulcer trouble may not be obtained but nevertheless on X ray considerable scarring and maybe pseudo diverticulum formation may be seen suggesting that they have had an ulcer for many years

RESPONSE TO TREATMENT —Another important factor to be considered when contemplating surgery is the patient s response to medical therapy Failure of adequate medical treatment or inability or unwillingness to accept the limitations implied thereby frequently necessitate operation Unfortunately the assessment of failure of adequate medical treatment ' is often difficult vague and unsatisfactory As mentioned in the opening paragraph of this chapter it is doubtful if any measures apart from rest and stopping smoking have any significant effect on the life history of a peptic ulcer (Jones 1957 Doll *et al* 1958) and medical regimes often receive the credit for remissions which have in fact occurred naturally So long as medical measures (or nature) relieve the patient s symptoms they should of course be persisted with A bald statement by a patient that medical treatment does not help him should not be accepted too readily the surgeon must satisfy himself that the physician s advice has been carried out properly and conscientiously for a reasonable length of time Nowadays there is an unfortunate tendency for patients who have not had medical treatment to come to the doctor and demand operation because a friend they know at work had a good result from surgery Such patients are likely to pay scant attention to any medical instructions and their response to surgery may be equally unsatisfactory At the other extreme once a peptic ulcer has penetrated into an adjacent structure such as the pancreas causing back pain it is improbable that anything the physician can do will induce the ulcer to heal Furthermore there is an increased risk of haemorrhage and other complications and for these reasons operation is advisable

AGE AND SEX —The age and the sex of the patient have some bearing on the choice of treatment With duodenal ulcer in men it was formerly thought that operation was inadvisable before the age of thirty as the results would be unsatisfactory but Capper and Welbourn (1955) found that age had no effect on the incidence of post gastrectomy syndromes However many patients in their twenties obtain rapid relief from medical measures and frequently their ulcers are of short duration so that surgery can at least be deferred There are unfortunately some young patients who develop symptoms before the age of twenty and soon experience some or all of the major complications of duodenal ulceration They frequently have a family history of ulcer trouble their response to medical treatment is short lived and surgery is soon required There is some

evidence that children may develop ulcers at times of stress such as examinations (Goldberg 1957) and in these patients operation should obviously be avoided in uncomplicated cases At the other extreme in old age surgery is being utilised increasingly with good results (Davey and O Donnell 1956) relieving old people from much suffering and the risk of complications which may the more readily prove fatal at their age As regards sex the proportion of female patients needing operation is less than it is in men Peptic ulceration appears to be a rather less virulent affliction in women than in men and is more amenable to medical treatment Furthermore the results of operation tend to be less satisfactory in women and in younger women iron deficiency anaemia is particularly common Surgery should therefore be postponed whenever possible till the menopause has been passed

Psychological and economic considerations —Many surgeons believe that a number of the poor results of gastrectomy are due to the operation being carried out in psychologically unsuitable individuals Emotional instability may be difficult to assess in the presence of an organic lesion and in general the benefit of the doubt must be given to the patient who may need to be relieved of genuine dyspeptic symptoms before the psychological overlay can be resolved Alcoholics are undoubtedly bad subjects for gastrectomy and the alcoholic aroma emanating from some patients with persistent post gastrectomy troubles can be most striking Unfortunately patients who are unable to control their imbibing tendencies are most unlikely to control their ulcers by medical treatment and surgery may be the lesser of two evils Such patients may in fact constitute an irreducible minimum of unsatisfactory results following any form of treatment

Economic considerations frequently complicate the decision about treatment Few men can afford to be off work on account of their ulcer for more than a few weeks each year and irregular attendance at work may well lose them their job Illingworth (1953) has however pointed out that a patient who is off work frequently may sometimes prove to be neurotic and conversely a patient with severe symptoms often continues at work If a patient s occupation is likely to involve his working in places where medical services are not available for example on board ship or on overseas exploration then it is safer to treat his ulcer by operation before he proceeds abroad

The cancer hazard —The factors which have been discussed above apply particularly to the decision for or against operation for duodenal ulceration Many of the factors also apply when considering patients with gastric ulcers although in this group the problem of the correct line of treatment is usually simpler Gastric ulcers and gastric cancers arise in the same type of mucosa the complications

of gastric ulcers carry a much higher mortality than do the corresponding ones due to duodenal ulceration and the results of surgery for gastric ulcer are good These three basic facts should take precedence over recondite argument about the site size and duration or the percentage error in diagnosis and risk of malignant degeneration in ulcers in the stomach Such factors may be of interest and value in considering large series of ulcers but all too often they are equivocal and unhelpful in the individual case In spite of radiology gastroscopy and exfoliative cytology errors in differentiating benign ulcers from ulcerated cancers still occur Most giant ulcers are chronic and benign (Jennings and Richardson 1954) and a large superficial benign ulcer —the so-called gastric bedsore—may develop in a few weeks In spite of the risk that some such ulcers may prove to be carcinomatous it is reasonable to treat any apparently benign gastric ulcer occurring in the usual site (that is on or within an inch of the lesser curvature down to the incisura) by medical means initially But if on full assessment by barium meal and gastroscopic examinations there is any reason to suspect malignancy or if the ulcer is found not to have healed or greatly improved after six to eight weeks or if it has recurred when checked three months later then that ulcer should be treated surgically Operation removes the risks of haemorrhage and perforation and of cancer developing in the atrophic mucosa chronically irritated by the ulcer On account of the possibility of the lesion being or subsequently giving rise to a cancer it is safer to operate on any gastric ulcer detected in a patient with histamine fast achlorhydria or full blown pernicious anaemia (Welch 1955)

About a fifth of ulcers in the pyloric region and one sixth of the rare ulcers on the greater curvature are cancers The risk of a lesion seen in these less common sites being malignant is so great that they should always be operated upon It is in fact a good working rule that any peptic ulcer occurring in an unusual situation should be treated surgically and not medically Such ulcers include those found in a hiatus hernia along the greater curvature or *in the pyloric region the second part of the duodenum the jejunum or beside* a Meckel s diverticulum

NUTRITIONAL FACTORS—The patient s general condition and nutrition must also be considered When a patient obeying medical instructions is persistently underweight or is losing weight his ulcer is probably not being fully controlled and surgery is likely to be a more satisfactory form of treatment It is unlikely however that much weight will be gained post operatively Sometimes an active ulcer is found in an obese patient Obesity renders surgery technically difficult and such patients must be given a chance to respond to less radical measures failing which an attempt is made to reduce their weight before

surgery is undertaken General systemic diseases usually contra indicate surgery but moderate degrees of benign hypertension (up to say 200 mm Hg systolic pressure) should not be allowed to affect the decision about treatment and in some otherwise unsuitable patients the risk of malignancy may become the paramount consideration Most patients with both active pulmonary tuberculosis and peptic ulcers should be treated medically Sometimes it is impossible to get sufficient nourishment into these patients to enable their lung lesions to heal and operation may be called for Gastrectomy may activate quiescent pulmonary tuberculosis (Pearson 1954 Krause 1957 Pulvertaft 1958) and should generally be avoided in tuberculous patients they can however be treated successfully by vagotomy and gastroenterostomy if other considerations necessitate operative intervention Asiatic patients whose diet consists largely of rice and spices frequently cannot afford the expense of medical regimes but like tuberculous patients they are liable to fare badly after the standard varieties of gastrectomy Rice is a bulky foodstuff and some of the patients are already on the borderline of nutritional inadequacy They are better treated by vagotomy and either gastro-enterostomy or pylorectomy (Stock *et al* 1956) which procedures leave an adequate gastric reservoir and are unlikely to precipitate nutritional deficiencies

In deciding which line of treatment to adopt the patient will always benefit if the physician the surgeon and the family doctor can discuss the case together The latter can supply useful information about the patient's mental make up and reactions his home circumstances and mode of living Every effort should be made to arrange such joint consultations

THE RATIONAL BASIS OF SURGERY

Ultimately the rationale of any operation is to be sought in the cause of the condition for the relief of which the operation is done Unfortunately in peptic ulceration the cause is still incompletely understood However in duodenal ulceration at least it is not disputed that acidity is a constant factor and that its control is a prime consideration In gastric ulcer it is not so easy to recognise the rational basis of the methods employed but the resection type of operation which has been found beneficial in duodenal ulceration has also proved satisfactory in the treatment of gastric ulceration

In the latter condition gastrectomy by decreasing what may be a normal gastric acidity may help to protect the atrophic and weakened mucosa from the attacking forces of acid and pepsin At the same time it removes the ulcer and that part of the stomach in which a new ulcer or a cancer is most likely to develop

The Reduction and neutralisation of acidity

The amount of acid produced by the stomach may be reduced in the following ways

1 By excision of the acid secreting cells These are situated mainly in the body and fundus of the stomach where resection is attended by maximal technical difficulty

2 By excision of the pyloric antrum which secretes gastrin the hormone which in turn stimulates the body and fundus to produce acid

3 By division of the vagus nerves which are secretomotor to the stomach

The gastric acidity may be neutralised in part or in whole by the introduction into the stomach of the alkaline juices mainly of the pancreas but also of the liver and duodenum

It is important to make the best use of our present knowledge without however taking an over simplified view of these mechanisms of acid secretion and neutralisation Recent work on the control mechanisms found both in the antrum and in the duodenum (Woodward *et al* 1954 Uvnas *et al* 1956 Sircus 1958 and others) has shown that in the experimental animal at least the gastric phase of gastric secretion and gastric motility may be both stimulated and inhibited by the pH and osmolarity of the chyme leaving the stomach and by the degree of distension of the antrum Exposure of antral or duodenal mucosa to a highly acid or hyperosmolar solution inhibits secretion as does antral distension but an alkaline reaction within the antrum (and possibly an acid reaction in the jejunum (Sircus 1953 1958)) stimulates the secretion of hydrochloric acid in the stomach

Thus various influences can act at several different sites some of them such as changes in acidity probably act through neural pathways while others may involve a humoral mechanism in turn the responsiveness of the antrum may depend in part on the integrity of the vagus nerves (Thal *et al* 1957) and numerous other inter relationships are possible Great caution *must of course always be exercised in applying experimental findings to* either physiological processes or pathological conditions in human subjects but the antrum is in the writer s opinion undoubtedly important in the stimulation of gastric secretion not only in the dog but also in man (Wells and Brewer 1948) although Stubbe (1957) from observations on antrectomised patients suggests that even this may not be so

NEUTRALISATION —The pancreas is the main source of antacid in the form of sodium bicarbonate and acid chyme in the duodenum is a powerful stimulus to pancreatic secretion Annis and Hallenbeck (1952) have shown that graded partial gastrectomy in the dog reduces pancreatic secretion proportionately and may delay its onset for some hours after a meal In their experiments

however the pancreas still responded normally to the instillation of acid into the duodenum and to injected secretin From studies on subjects with normal gastro intestinal anatomy Atkinson and Henley (1955) concluded that intra duodenal pH was more dependent on gastric secretion than on biliary and pancreatic factors

There can be no doubt however about the importance of the neutralising powers of the pancreatic and biliary secretions in the prevention of jejunal ulceration after a gastro jejunal anastomosis has been made The intrinsic resistance of the jejunum to ulceration is less than that of the duodenum and so to prevent ulcers developing in the jejunum after a gastro jejunal anastomosis either its mucosa must be bathed in alkaline juice or gastric acidity must be markedly and permanently decreased

THE ANTRUM AND GASTRIN —In the so-called physiological gastrectomy in which the antrum is left in continuity with the duodenum the reaction within the antrum becomes highly alkaline and causes the release of gastrin which stimulates the remaining acid secreting part of the stomach and so gives rise to jejunal ulceration (Wells and Brewer 1948) Wangensteen (1952) in his segmental removal of eighty per cent of the stomach performs a pyloroplasty and leaves the denervated antrum which is joined to the small proximal gastric fragment He believes that so little acid secreting tissue remains that any gastric stimulation from the antrum can do no harm His results confirm this belief (Wangensteen 1957)

After gastro enterostomy alone or with vagotomy if the stoma is not in the most dependent position and especially if it is made too far to the left alkaline secretions entering the stomach may collect in its distal atonic portion and stimulate the release of gastrin This mechanism may help explain why stomal ulceration is relieved by vagotomy with less certainty when it complicates a gastro enterostomy than when it follows a partial gastrectomy

Dragstedt (1958 1959) believes that many gastric ulcers are the result of secretion being stimulated by the release of gastrin in the pyloric antrum The shape of the stomach too may play a part in the development of a gastric ulcer It has been noticed that many gastric ulcers occur in the elongated hypotonic type of stomach (Johnson 1957) In a stomach of this type acid from the proximal part trickles undiluted down over the mucosa of the elongated body of the stomach any alkaline juices present remaining in the region of the antrum Gastrectomy corrects the shape of this type of stomach and at the same time removes the antrum and part of the mucosa which secretes acid and which may be prone to further ulceration or neoplastic change Reconstruction by some modification of the Billroth I gastro-duodenal anastomosis is usually adopted since in gastric ulcer cases general nutrition tends

to be poor and the routing of food through the duodenum favours absorption Vagotomy is unnecessary and anastomotic ulceration does not occur

ACID SECRETING MUCOSA —Resection of acid secreting gastric mucosa is an integral part of every gastrectomy and it may be taken as axiomatic that *in the average case provided the antrum is removed the higher the level of* gastric section the greater the fall in the output of hydrochloric acid and pepsin Total gastrectomy deprives the alimentary canal of all hydrochloric acid

As this truth became apparent surgeons began to be more radical in their resections of the stomach the limit being reached probably by Visick (1948) who reported a series of subtotal resections using the Polya procedure with virtually complete exclusion of jejunal ulceration Visick s plan was widely adopted jejunal ulcer ceased to be a cause for anxiety but the occur rence *of post gastrectomy syndromes soon became apparent*

It is pertinent to ask whether it may not be possible to forecast the level at which the stomach may need to be resected in any individual case and the reader is referred back to page 28 where the total mass of acid secreting cells is discussed It will be seen however that gastric secretion is influenced by other factors as well as by the total parietal cell mass which is not in itself therefore a sufficient guide to the type and extent of operation called for in the individual case Walton (1950) and Taylor (1944) using test meal records radiological and gastroscopic appearances tried for years to select suitable cases for gastro-enterostomy and others for gastrectomy on the basis of their capacity for secretion The method did not gain general acceptance and since it is no longer in routine use it may be assumed to have proved disappointing in the long run Bruce *et al* (1959) (see p 92) are exploring the use of maximal histamine stimulation of secretion as a guide to the best type of operation

Numerous attempts have been made to popularise the notion of resecting the acid bearing *portion of the stomach* (Wangensteen 1952 Deloyers 1956) whilst conserving a reasonable gastric reservoir but the technical difficulties are such that these are unlikely ever to prove generally acceptable procedures

THE VAGUS NERVE —Vagotomy is the other approach to this problem The method was first suggested for and used in clinical practice by Edwards (1939) on the basis of experimental work in animals carried out in the Department of Surgery in Liverpool Unfortunately his operative technique was imperfect and the procedure fell into disrepute until 1943 when Dragstedt revived it so brilli antly and successfully It is surprising that after twenty years so much mystery

still surrounds the early and late physiological results of this procedure The following statements represent the writer s views at the time of writing and will probably not be seriously disputed

1 Complete vagotomy is usually possible *via* the abdomen and practically always across the chest In the event of either approach proving only partially successful the addition of the other as a secondary procedure is likely *to be wholly* rewarding Twenty five per cent of abdominal vagotomies are said to be incomplete

2 Complete vagotomy causes gastric atony and complete or nearly complete achlorhydria

3 Some degree of gastric tone and motility returns within three months to a year

4 Achlorhydria may persist—presumably indefinitely

5 After an interval of some months acid may reappear in the stomach in the following circumstances

(*a*) If the antrum has not been removed gastrin may stimulate secretion (*Woodward 1958*)

(*b*) If the antrum has been removed but the night secretion continues at or above normal levels it must be assumed that either the vagal operation has been incomplete or that regeneration has occurred

6 Intestinal disturbances such as diarrhoea occur after gastrectomy of any sort but seem to be commoner after vagotomy The explanation is obscure and the diarrhoea usually settles within six to twelve months The occurrence of this phenomenon may not be *related to vagotomy the point is unproven* Numerous studies in Liverpool on intestinal motility after operations suggest that vagal resection neither hastens nor retards the progress of the bowel content Burge and Clark (1959) believe that inhibition of pancreatic secretion after vagal section causes impaired digestion of fat with consequent steatorrhoea and intestinal hurry They advocate selective vagotomy sparing the branch of the posterior vagus which goes to the coeliac plexus

Vagotomy has certainly come to stay and is a most valuable aid to the reduction of gastric acidity Its availability has increased the complexity of the problem of choice of operation by bringing back for consideration such arrangements as gastro enterostomy and the anastomosis-en Y of Roux (1897) both of which had been discarded because of their *high rate of jejunal* ulceration

Discussion

GASTRO-ENTEROSTOMY —For about forty years up till 1939 gastro-enterostomy was the operation most commonly performed in Britain

for the relief of peptic ulceration After 1945 it was rapidly supplanted by the Polya* type of partial gastrectomy and by 1950 the Billroth I* operation was being tried out in many centres Both these types of gastrectomy had however been in common use for more than a quarter of a century in the surgical clinics of Central and Northern Europe Both of them have given satisfactory results in eighty to ninety five per cent of cases in the remaining five to twenty per cent of cases the results have been unsatisfactory on account of recurrent ulceration or severe post gastrectomy syndromes In consequence during the last ten to fifteen years a number of different operative procedures have been devised and tried in an attempt to find the ideal surgical treatment for all peptic ulcers Unfortunately this ideal method has not yet been found and the multiplicity of available operations has confused rather than clarified the surgeon s task in choosing the best procedure for the individual case Five to ten year results are not yet available for many of the newer techniques and even for the older operations more really long term follow up studies such as those of Wells and MacPhee (1956) and Krause (1957) are necessary before the best treatment for various types of peptic ulcer can be finally determined Controlled studies are also urgently needed

Gastro-enterostomy alone is no longer done for either gastric or duodenal ulcers It cannot affect the natural history of a gastric ulcer When it is used to treat a duodenal ulcer a recurrence develops in up to forty per cent of patients (Clark 1951) which is most unfortunate since in patients lucky enough to escape recurrent ulceration the results are really excellent and post gastrectomy syndromes are rare Why sixty per cent of patients had such good results has never been explained In pyloric stenosis the severe ulcerogenic tendency responsible may be temporarily masked by the effects of gastric retention but once the stomach is drained by a simple gastro enterostomy the abnormally great digestive forces become active again and soon cause recurrent ulceration *Pyloric stenosis requires the same radical forms of surgery as does any other severe duodenal ulcer*

GASTRIC RESECTION AND SHORT-CIRCUIT COMPARED —Intractable duodenal ulceration is the most common indication for surgery in peptic ulcer disease and the Polya gastrectomy is the operation which has been most carefully studied and followed up At the present time the principal alternatives to it in the treatment of duodenal ulcer are the Billroth I gastrectomy and vagotomy plus gastro-enterostomy The operative mortality is nearly the same one to three per cent for each of the three

*The term Polya gastrectomy here denotes any type of gastrectomy with a gastro jejunal anastomosis while Billroth I refers to any type of gastrectomy with a gastro duodenal anastomosis

procedures (Anderson *et al* 1955 Kanar *et al* 1956 MacKelvie 1957)
being only slightly less for vagotomy plus gastro-enterostomy than for the
two types of gastrectomy Each of the operations gives good or satisfactory
results in eighty to ninety five per cent of cases the patient s assessment of
the result often being more favourable than that of the doctor These three
procedures must be analysed in relation to their effects on the patient s
nutrition and on the incidence of the post gastrectomy syndromes and of
recurrent ulceration Unfortunately different surgeons have reported widely
different results when using the same operation

RECURRENT ULCERATION AND POST GASTRECTOMY SYNDROMES —The
incidence of recurrent ulcer proved and suspected after a Polya gastrec
tomy for duodenal ulcer when seventy five per cent of the stomach has
been removed is from two to six per cent (Capper and Welbourn 1955
Anderson *et al* 1955 Walters and Lynn 1957) or even less (Wells and
Brewer 1948) With a Billroth I operation however the recurrence rate
usually is from ten to twenty per cent (Goligher *et al* 1956 Wallensten
1957 Walters *et al* 1957) This high recurrence rate makes the Billroth I
operation an unsuitable method for treating duodenal ulceration unless a
vagotomy is carried out at the same time

With vagotomy plus gastro enterostomy the reported recurrence rates
vary from one per cent (Holt and Robinson 1955 Hindmarsh 1957) to
seven to fifteen per cent (Bennett Jones and O Domhnaill 1955 Henson and
Rob 1955 Everson *et al* 1957 Walters and Mobley 1957)

Although recurrent ulceration undoubtedly constitutes the greatest threat
to life the other undesirable sequelae may be very distressing to the patient
and loss of weight may lower resistance to pulmonary tuberculosis Setting
aside for the moment the Billroth I procedure the Polya operation carries
a lower recurrence rate but a higher incidence of severe post gastrectomy
syndromes including nutritional deficiencies than vagotomy plus gastro-
enterostomy With the Polya operation the higher the resection the lower
the recurrence rate (Capper and Welbourn 1955 Pulvertaft 1958) but the
greater the tendency to loss of weight (Welbourn 1953) vitamin deficiencies
(Blake and Rechnitzer 1953) dumping and bilious vomiting (Wells and
MacPhee 1952) The exact type of anastomosis—whether antecolic or
retrocolic afferent loop to greater or to lesser curvature with or without
a valve—is generally believed to have little effect on the incidence of these
various syndromes Taylor (1959) however thinks that many of the ill effects
of gastrectomy are due to alterations in the anatomical relationships around
the duodeno-jejunal flexure For some years he has fashioned a tubular gastric
stump and brought it down behind the colon to anastomose it to the jejunum
just beside the flexure The results of this procedure are very satisfactory

To return now to Billroth I it is commonly believed that the post gastrectomy syndromes are to be attributed for the most part to the Polya arrangement and this had been the writer s own view but it may well be that this relationship is more apparent than real and that it arises from the preponderance of Polya gastrectomies in duodenal ulcer surgery There is relatively little evidence available about these syndromes after Billroth I type procedures which in hyperchlorhydric patients lead to such a high incidence of anastomotic ulcer that this latter complication has quite obscured the other issue Moreover the combination of vagotomy with gastro-duodenal anastomosis has received little attention to date in post gastrectomy studies In the writer s series of eighty nine such cases of vagotomy in which a hemi gastrectomy was done and an unusually wide gastro-duodenal stoma made no less than six patients had quite severe dumping and almost as many as would have been expected in a similar series of Polya gastrectomies had milder symptoms of a similar character

It would seem that the importance of the afferent loop in the aetiology of dumping must therefore be thought about again and more attention paid to the rival claims of intestinal hurry and diminution in plasma volume resulting from the too speedy outpouring of excessive quantities of digestive juices and intestinal transudate (see Chapter XII)

Others have found the early results of vagotomy plus hemigastrectomy with gastro-duodenal anastomosis good with dumping less frequent and recurrence no more frequent than with a seventy five per cent Polya gastrectomy (Johnson and Orr 1954 Edwards and Herrington 1957) With the wider type of gastro-duodenal anastomosis and hemigastrectomy described by the writer (Wells 1957) (see p 115) the general results were as has been said less satisfactory and three of the eighty nine patients had proven and two had suspected recurrent ulcers when reviewed one to four years after operation These however were in every case relieved by transthoracic vagotomy and were certainly all due to incomplete abdominal section of these nerves The need for careful exposure and thorough resection of the vagi is a lesson which should be learned by precept rather than by repeating old mistakes! Recently in a series of patients with duodenal ulcers the writer has been using vagotomy plus antrectomy only (thus leaving a larger gastric reservoir) with a wide gastro-duodenal anastomosis and no case of dumping has so far been encountered When a gastro-jejunal anastomosis is employed vagotomy plus pylorectomy gives very few post gastrectomy syndromes (Stock *et al* 1956) and vagotomy plus hemigastrectomy has also shown a low incidence of such syndromes and of recurrent ulceration (Farmer *et al* 1951 Smithwick 1957)

The anastomosis-en Y of Roux (1897) allowed the gastric acid to enter the jejunum without there being any alkali available there to neutralise it and was abandoned because of jejunal ulceration Recently it has been revived as an elective procedure in duodenal ulcer surgery jejunal ulceration being guarded against either by the performance of a very high gastrectomy (Schofield and Anderson 1953) or by doing an abdominal vagotomy (Gow 1958 personal communication) In a series of 162 patients treated by very high gastrectomy and Roux Y anastomosis and followed up for from one to six years there was only one recurrent ulcer (Schofield and Denton 1959) The early results of vagotomy, partial gastrectomy and Roux Y anastomosis are also good (Gow 1958 personal communication) Others including the writer (Wells and Johnston 1955) have used this technique in selected cases of hiatus hernia and the afferent loop syndrome It is especially useful in cases of cardio oesophageal incompetence because regurgitant vomiting is prevented by the rapid emptying of the stomach and the fact that it never contains any bile Fears that the early post cibal syndrome might be troublesome and that impaired nutrition would be a common feature have not been realised

The great advantage in vagotomy plus gastro enterostomy lies in the fact that the gastric reservoir remains intact and so the operation causes relatively little interference with the patient s nutrition (Lloyd Davies 1956 MacKelvie 1957 Cox and Kerr 1957) many of the patients put on weight after operation and there is less tendency to anaemia than after a Polya resection (Smart and Williams 1958 Burge and Pick 1958) The operation may therefore be of especial value in premenopausal women and in men in whom the maintenance of adequate nutrition is of more than usual importance such as those with quiescent pulmonary tuberculosis (Lloyd Davies 1956) A two thirds Polya gastrectomy or a vagotomy plus gastro enterostomy is used by Bruce *et al* (1959) when Kay s augmented histamine test (p 64) shows the acid concentration is low for patients with a high acidity (over 50 mEq /hour) he performs a two thirds gastrectomy plus vagotomy Kay (1959 personal communication) also suggests that the efficacy or otherwise of vagotomy in controlling hyperacidity can be estimated before operation by medical vagotomy in which the autonomic nerves are blocked with hexamethonium and atropine before the augmented histamine test is carried out

Vagotomy plus pyloroplasty appears to be slightly less satisfactory than vagotomy plus gastro-enterostomy in the treatment of duodenal ulcers (Lloyd Davies 1956 Hindmarsh 1957) However by combining vagotomy with a local excision of the anterior part of the pyloric sphincter Holt (1959 personal communication) has obtained good results Vagotomy alone (Edwards 1939, Dragstedt 1943) is nowadays never performed since gastric ulcers develop in

a quarter of the patients so treated (Slaney *et al* 1956 Walters and Mobley 1957) and foul eructations and diarrhoea may be troublesome some type of drainage procedure must be carried out at the same time

VAGOTOMY —In any of these operations in which the control of gastric acidity depends on vagotomy it is important that the vagotomy should be complete There is however some recent evidence which suggests that division of the vagal fibres to the coeliac plexus may cause pancreatic insufficiency and diarrhoea (Burge and Clarke 1959) It is not always possible to be sure that all nerve fibres to the stomach have been divided indeed there is always some degree of presumption in this procedure If even one or two strands are left the stomach may still respond to vagal stimulation (Burge and Vane 1958) and since autonomic nerve fibres can sprout (Murray and Thompson 1957) in time there may be an important degree of recovery of gastric secretion Burge and Vane (1958) have described a test for assessing the completeness of vagotomy by recording intragastric pressure when applying stimulating electrodes to the abdominal oesophagus It is doubtful whether this test is suitable for general application in its present form but some simplified development of it may prove useful

TOTAL GASTRECTOMY —Total gastrectomy has no place in the routine treatment of benign ulcer but may be done in certain circumstances *e g* when a gastric ulcer is mistaken for carcinoma when ulceration continues in spite of the removal of a non beta cell tumour of the pancreas or when for no determinable reason anastomotic ulceration persists in spite of high gastric resection and reliable vagotomy Such cases must indeed be rarities and it would be misleading to presume to generalise about them It can however be said that total gastrectomy is a rather crippling operation usually involving one or more of the following handicaps *viz* loss of appetite loss of weight reflux oesophagitis diarrhoea steatorrhoea and post prandial distress Anaemia is avoided only by the administration of iron and vitamin B_{12} or whole stomach extract When however total gastrectomy has been performed for a benign lesion the patient may afterwards maintain reasonable health and lead a useful life (Kyle 1959 personal communication)

Of the available techniques Balint and Gumner (1958) suggest that anastomosis by Roux-en Y has no disadvantages and is the least likely to be complicated by reflux oesophagitis This conclusion is in accord with the writer s own experience the Mayo Clinic workers however rather favour a continuous jejunal loop with entero anastomosis (Fly *et al* 1958)

OTHER PROCEDURES —Of the many other operations devised during the last ten to fifteen years for the treatment of duodenal ulcer none has as yet been followed for a sufficient length of time for a final opinion to be formed about its

merits A few have however rapidly proved themselves unsatisfactory Antro-duodenectomy and deep X ray therapy to the stomach (Brown *et al* 1952) failed to control gastric secretion and by the end of two and a half years twelve per cent of the patients so treated had suffered a recurrence (Brown and Wood 1956) Attempts to increase the extent of the gastric resection in a Billroth I operation by inserting a length of jejunum (Henley 1952) or trans verse colon (Moroney 1951) between the gastric stump and duodenum were unsuccessful on account of the high incidence of recurrent ulceration The colonic implant protected by the addition of abdominal vagotomy is however of value in the treatment of certain selected cases of severe dumping symptoms Although in the hands of its modern advocate segmental gastric resection plus pyloroplasty carries a low operative mortality (Wangensteen 1952) in less skilled hands the mortality rate is likely to be higher For the same reason proximal gastrectomy plus pyloroplasty (Deloyers 1956 Millbourn 1957) cannot be recommended in the routine treatment of peptic ulceration

The relative value of directing the food stream through the duodenum is theoretical remains unproven and is likely to remain unproven unless and until further contrasted series of operations based on random selection are available for study Meantime surgeons can rest assured that the Polya and with vagotomy added Billroth I gastro jejunostomy and even Roux-en Y all afford excellent and rewarding results Partial pylorectomy has been used for drainage after vagotomy (Beattie 1950) and is being used bv Holt (1959 per sonal communication) but in the writer s view may lead to pyloric stenosis or stimulation of the antrum to secrete gastrin

Summary

It remains then for each surgeon to decide for himself and in the circum stances in which he finds himself the operation which he will of choice routinely employ in uncomplicated duodenal ulceration

The beginner would be well advised to adopt vagotomy and gastro enterostomy to be unhurried careful and thorough Many because of their training and the practice of those with whom they have worked will want to follow Polya These should exercise extreme caution when faced with a difficult duodenum for the greatest drawback to this procedure is the inevita bility sooner or later of a fatal blow-out of the duodenal stump or a disastrous leak from the pancreas

Uncomplicated gastric ulcer is best dealt with by hemigastrectomy with excision of the ulcer and gastro-duodenal reanastomosis Gastric ulcers associated with duodenal ulcers are usually associated with hyperchlorhydria (Johnson 1957) and are best treated as for duodenal ulcer

Those who like the present writer feel that the last word has not yet been said and that it is necessary to try such unproved methods as commend themselves for further study should do two things they should test their new choice against their previous favourite operation by a method of random selection* and they should institute a reliable system of follow up by personal interview by a team of impartial observers with some sort of continuity so that the standard remains reasonably constant Only thus will progress be made The writer s current studies on this basis in duodenal ulcer surgery incline him to regard as the operation of choice vagotomy resection of the antrum with any non adherent ulcer and gastro-duodenal anastomosis (Bill roth I) Penetrating ulcers are *not* resected The operative technique is described in the following chapter

* Random selection means selection on a basis wholly unrelated to the peculiarities of the individual case Alternation is unsatisfactory because it is open to abuse by subconscious bias Odd and even dates of birthdays the drawing of a marked card these and similar devices subject only to the laws of chance are the only safe method of randomising

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CHAPTER VI

THE OPERATIVE TREATMENT OF PEPTIC ULCERATION

By Charles Wells

Preparation for operation

MUCH can be done to improve the general condition of those patients who because of malnutrition are anaemic or dehydrated or whose level of plasma protein is seriously low Much of the technical difficulty that arises during partial gastrectomy is due to active periduodenal or perigastric inflammation and oedema A course of the strictest medical treatment in hospital and with abstention from smoking can convert the most formidable ulcer into one that can be tackled without too much difficulty Repeated aspiration of the stomach is beneficial if there is delay in its emptying and in patients with very active ulceration and severe symptoms continuous intra gastric feeding may be necessary

In all cases there is everything to gain by at least one week s treatment in which fluid protein haemoglobin and vitamin deficiences are made good Except in emergencies gastrectomy should not be performed unless the patient s haemoglobin is more than ninety per cent For patients with chronic bronchitis the time can be usefully spent in improving the respiratory efficiency by graduated breathing exercises In all deliberate operative work the anaesthetist is given an opportunity to advise in the preparations just as he is expected to show responsibility in the after-care Unless there are clear and definite indications the administration of antibiotics should be avoided as they often play a part in the aetiology of post operative enterocolitis pneumonia and other antibiotic resistant bacterial invasions

Obesity is not unusual in patients suffering from peptic ulceration and may seriously increase the technical difficulty of gastrectomy The rigid exclusion of mainly carbohydrate foodstuffs (sugar bread sweets pastry buns scones and potatoes) from the diet will almost certainly reduce the body fat significantly and usefully within a period of two to three months or less This type of diet is however completely different from that usually prescribed in the medical treatment of peptic ulcer and will not help to reduce the activity of the ulcer (Kemp 1958 personal communication) On the other hand the efficacy of ulcer diets has been questioned and rest both bodily and mental is probably the most effective single factor in medical treatment The writer in a limited experience has had no reason to regret the prescrip tion of a very low carbohydrate diet in obese ulcer patients

The majority of patients are not obese and may have lost weight Unfortunately among the general public one of the criteria of the success of any abdominal operation is that the patient should rapidly put on weight afterwards If a gastrectomy is contemplated it is therefore worthwhile in order to avoid disappointment to tell the patient that he is unlikely to put on much weight He should also be told that for a few months after operation *he may find that his stomach will not comfortably hold a large meal* Apart from these two gentle warnings the patient should and can rightly be encouraged to believe that once he has had his operation he will be able to eat work and live perfectly normally once again

Blood is grouped and cross matched although transfusion is seldom needed in routine gastric surgery

Gastric lavage is necessary only in cases of pyloric stenosis but a Ryle s tube is usually passed into the stomach and fixed externally before the patient goes to the theatre Alternatively whatever type of post-operative naso-gastric tube is selected by the surgeon can be introduced on the table by the anaesthetist and accurately sited by the surgeon before closing the abdomen

OPERATIVE TECHNIQUE

It is not the writer s intention to give a complete detailed and fully illustrated description of the operations which may be used in treating peptic ulcers Many such descriptions already exist and in any case the correct place to learn the technique of an operation is in the operating theatre assisting a *surgeon skilled in its performance In the following pages only an outline* of the most useful and usual operations is given but emphasis is laid on practical points and on methods of overcoming difficulties Procedures which have become standard in the treatment of post gastrectomy syndromes and recurrent ulceration are described but for a more detailed consideration of the surgical management of these conditions the reader is referred to Chapters XII and XIII

Incision and exploration

Whatever approach is used should give ample access which for median and *paramedian incisions* means extension from the umbilicus to the angle between the xiphisternum and the costal margin Excision of the xiphisternum is a useful addition on occasion and in case of real difficulty the sternum itself may be split upwards in the midline and then outwards into the 8th or 7th left interspace

Within the peritoneal cavity a systematic examination of the other viscera having been made attention is directed to the duodenum and stomach

Duodenal ulceration may be immediately obvious or may be detected only with some difficulty Chronic ulceration fixes and *shortens* the duodenum —a very characteristic finding Antero lateral ulceration causes Cayenne' stippling of the overlying peritoneum even when there is no gross deformity If necessary the duodenum can be mobilised by making a curved incision lateral to it and lifting it forward together with the head of the pancreas Finally and extremely rarely it may be necessary to incise the duodenal wall and explore its interior

In the stomach again an ulcer may be immediately obvious or the surgeon may take a long while to satisfy himself that no ulcer is present Radiological and gastroscopic findings suggest the site at which the lesion is to be expected The lesser sac should be opened if in doubt Opening the stomach to look for an ulcer is not really rewarding in elective surgery although it may be necessary in emergency surgery for haemorrhage

The oesophageal hiatus of the diaphragm is examined in every case so that the operator may become familiar with the normal and be able to recognise herniation when it is present and form an opinion about its degree Normally the peritoneum overlying the hiatus forms a smooth firm sheet but in herniation it can be telescoped upwards through the hiatus into the chest with part of the stomach for half an inch or more up to say two or three inches in an extreme case At the same time the increased width of the hiatus can be appreciated Hiatus hernia is so important a cause of failure of the routine surgical treatment of duodenal ulcer because of post operative regurgitation of bile and/or food that its recognition is of first-class importance However severe oesophagitis may occur in very slight degrees of cardio-oesophageal retraction and quite marked herniation may be present without symptoms It is therefore of the utmost importance to pay heed to the relevant points in the history so that the significance of any operative findings can be correctly interpreted and treated

Next if the surgeon is minded to perform a gastrectomy with duodenal closure he must examine the duodenum to see whether the case is suitable for this procedure Caution is dictated by a large oedematous mass over lying the pancreatic head or by a grossly shortened duodenum especially when associated with an ulcer penetrating into the head of the pancreas Kyle (1958) has reported four cases of fistula into the common duct An unexpectedly trivial antero-lateral ulcer should always be given a second look to see whether there is not a second hidden ulcer on the pancreatic surface

Whenever there is any doubt about the wisdom of completing the intended operation the plan should be changed and an alternative procedure followed

NOTES ON OPERATIVE TECHNIQUES

Vagotomy

ABDOMINAL VAGOTOMY —The incision used must give good access to the left upper abdomen As a first step hiatus hernia is excluded by running a finger around the peritoneum overlying the cardia A gross hernia sometimes though by no means always makes abdominal vagotomy impossible In a stout or thick set individual the operation may be very difficult and the writer advises that if difficulty is experienced a secondary operation should be carried out through the chest immediately or on another occasion

Access is improved by giving the table a head up tilt and inclining it so as to raise the patient s left flank The left lobe of the liver is mobilised by incising its triangular peritoneal ligament With this lobe retracted to the right and with the stomach drawn down the peritoneum over the cardio-oesophageal junction is seen and incised The lower oesophagus is freed in the chest for a distance of about two inches by blind finger dissection A soft tube is then slipped around the oesophagus and used to draw it down two inches into the abdomen Alternatively a tube or corrugated rubber sling may be passed over the cardio-oesophageal angle from the front and brought down and out across the lesser sac and through an opening made in the gastro hepatic omentum (Tanner 1954) This type of sling disturbs the region traversed by the right vagus rather less and so may enable the nerve to be picked up more easily

Under vision the left (anterior) vagus is sought and divided where it lies in close apposition with the anterior aspect of the oesophagus If it cannot be seen it may be palpated as a tense cord If the oesophagus has not been drawn down far enough a single trunk may not be visible and the small branches may be hard to find The right (posterior) vagus may be seen in the loose tissue to the right of the oesophagus It is *not* under tension because the pull on the gullet draws down its upper attachment whilst its lower part is not incorporated in that part of the stomach on which traction is made At times it may be found more easily by the finger It tends to lie well away from the oesophagus itself backwards and towards the right and the inexperienced operator must remember that it is flaccid not tense It may be easiest to find it with the finger behind the oesophagus from the *left* side In this case it is hooked forward and to the left and so brought into view for resection It is essential throughout these procedures to handle the tissues gently since the musculature of the oesophagus and even its mucosal lining can easily be torn

After division of the two main nerve trunks additional branches must always be sought and dealt with In all cases the beginner should resect half

an inch of each nerve and of its branches between pairs of ligatures and have the tissue examined microscopically for confirmation The experienced operator may destroy a sufficient length of nerve tissue by diathermy applied between the blades of a pair of forceps (Fig 19) The forceps are necessary in order to conduct away the heat generated which might otherwise damage the oesophageal wall and lead to a delayed perforation

After abdominal vagotomy the cardio oesophageal angle should always be carefully restored by suturing The repaired area should then be anchored to the under surface of the diaphragm and the oesophageal hiatus narrowed if it is unduly patent These steps are necessary to guard against the not infrequent development of cardio-oesophageal incompetence after vagotomy The peritoneum should be carefully repaired to avoid leaving a raw area to which the intestine may become adherent

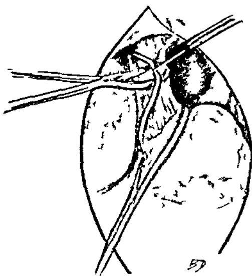


FIG 19
Destroying a short length of the right vagus nerve with diathermy The cardia is being drawn downwards by a catheter passed around it

TRANSTHORACIC VAGOTOMY —The left chest is opened by resecting a length of the 8th rib or opening the 8th space The lung is retracted to expose the aorta beyond which the oesophagus is found under the covering parietal pleura The inferior pulmonary ligament which usually contains a small vessel may have to be partly divided The pleura is incised and the oesophagus freed until a sling of Paul s tubing or other suitable material can be passed beneath it The oesophagus is then drawn well up into the cavity of the chest so that its circumference can be examined and the vagi identified and divided as has been described above

Ordinarily this is a very simple and pretty operation which however can be difficult in two sets of circumstances First if the pleura is adherent its dissection may be tedious but can usually be achieved with patience and care Second in hiatus hernia the oesophagus may be bound down by an inflammatory reaction and the vagi may then be difficult to find In such

a case it is wise to open the chest through the bed of the seventh rib in order to gain access to the oesophagus at a higher level

NOTES ON VAGOTOMY —1 A drainage operation on the stomach is always necessary

2 When the operation is done for the relief of jejunal ulcer the abdominal approach permits visualisation of the lesion but may prove difficult or even impossible of completion Moreover the ulcer may perforate or the surgeon may be tempted to embark on an unnecessary and difficult resection The thoracic approach is safe and certain and in the writer s view preferable unless there is some special reason for wishing to explore the lesion

3 Selective vagotomy (Burge and Clark 1959) is discussed on p 88

Posterior gastro-enterostomy

With the exposed stomach lying as nearly as possible in its natural position a tissue forceps is applied to the lowest point of the greater curvature and a second one opposite to it on the lesser curvature These presently serve as guides

The omentum and transverse colon are next brought forwards and upwards to expose the transverse mesocolon which is incised from beneath between the middle and left colic vessels to expose the posterior wall of the stomach along the line already selected from in front This portion of the posterior stomach wall is drawn through the mesocolon and picked up with tissue forceps opposite those previously placed anteriorly which are now removed A gastro-enterostomy clamp is applied to the portion of the posterior wall of the stomach thus selected The duodeno-jejunal flexure is then identified and a second clamp applied to a three inch length of jejunum almost immediately below the flexure The anastomosis is carried out on the surface of the abdomen which necessitates a certain length in the afferent loop This is quite sufficient to guard against tension causing angulation at the stoma Care is required not to allow the centre of the loop to come completely through the clamp (Fig 20)

The clamps are laid parallel to each other so that the proximal end of the jejunal loop is at the lesser curve which on account of the rotation upwards of the stomach and colon is now the lower point on the stomach wall The jejunum and stomach are then anastomosed in the usual two layers For the anterior inner layer a simple through and through or a Schmieden stitch is safer than an inverting Connell stitch which is not sufficiently haemostatic Since no nutrient vessels to the stomach are ligated as they are in gastrectomy it is wise after incising the sero-muscular coats to underrun and tie the exposed vessels before incision of the mucosa When the posterior inner

layer of the stitches has been inserted the clamps may be temporarily slackened to enable any persistently bleeding points to be seen and underrun

At the conclusion of the anastomosis the mesocolon is attached to the posterior wall of the stomach around the new stoma leaving the anastomosis in the greater sac The omentum and transverse colon are then returned to the abdomen leaving the suture line lying vertically and posterior

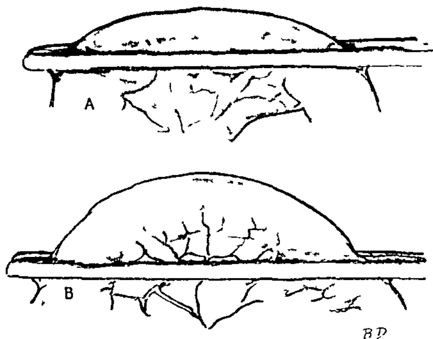


FIG 20
(A) Correct method of applying half of a gastro enterostomy clamp to the jejunum (B) Incorrect method—the blood supply to the intestinal wall is not properly controlled

The operation described is the standard no-loop posterior gastro-enterostomy of Mayo (1905) When used in conjunction with vagotomy special care must be taken to ensure that the stoma shall lie at the lowest point of the atonic stomach For this reason some surgeons prefer a horizontal isoperistaltic stoma running along the posterior surface of the stomach just behind the greater curve on the assumption that this ensures drainage of the dependent portion of the stomach with greater certainty An afferent loop of about three inches is appropriate A high stoma or one too far to the left must be avoided

Anterior gastro-enterostomy is occasionally necessary for technical reasons and the jejunal loop must then be long enough to encircle the omentum and transverse colon Alternatively it may be brought up through the great

omentum or through the mesocolon and gastro-colic omentum to reach the anterior wall of the stomach The stoma is usually made horizontally along the greater curve from left to right The procedure should be avoided if at all possible in non malignant ulceration

Antecolic Polya gastrectomy

The first step is to clear the greater curve of the stomach by dividing the short nutrient vessels running between it and the gastro-epiploic arch A beginning is made a little to the left of the lowest point of the greater curve When the lesser sac is freely open and the operator is proceeding towards the pylorus the posterior wall of the stomach is cleared of adhesion between itself and the transverse mesocolon with the special object of carrying the middle colic vessels safely away from the posterior stomach wall The remaining nutrient vessels are then divided until the proximal end of the duodenum is reached and until a number of the short vessels between the fundus and the spleen have been disposed of The whole of the gastro-epiploic arch the blood supply of the omentum is thus left intact

Not all surgeons believe this technique necessary It is simpler and quicker to apply more massive ligatures to the great omentum and in so doing to sacrifice its blood supply How important it is to leave the blood supply to the omentum intact is a matter of opinion and one about which there seems singularly little information to be gleaned either from the literature or from personal conversations



FIG 21
The hepatic artery may be inadvertently drawn up and ligated when the right gastric artery is being ligated and divided

The lesser curve is now cleared The right gastric artery is ligated and divided distal to the pylorus being careful to avoid damaging the main hepatic artery (Fig 21) Another small vessel running to the superior surface of the duodenum has often to be dealt with as well The lesser omentum is then divided (usually without further bleeding) to expose the left gastric vessels These important vessels are often obscured by fatty tissue and when there is difficulty in locating them it is sometimes easier to postpone clearing and tying them until after the duodenum has been divided and the stomach has been reflected over to the left Conversely if the duodenum is to be closed it may be easier to complete this step after the stomach has been divided and anastomosed to the jejunum and so got out of the way Once the left gastric vessels are cleared they are securely ligated

and divided between ligatures some surgeons like to put two ligatures on their proximal end Division of the vessels frees the lesser curve from its main anchorage and this part of the operation is completed by removing the vascular fatty tissue from about an inch of the lesser curve so as to leave it clean and ready for division and anastomosis

The next step is to dissect the first part of the duodenum including the ulcer cleanly away from the head of the pancreas until ideally three quarters of an inch of duodenum beyond the ulcer is exposed When mobilising the duodenum numerous fine strands of tissue are found joining its posterior and inferior surfaces to the adjacent pancreas Nearly all contain small vessels and they should be divided between pairs of fine haemostats and tied A sharp lookout should be kept for any accessory pancreatic ducts If any are found they should be carefully preserved but if one has been inadvertently divided the ends should be picked up and ligated with linen thread When the first part of the duodenum has been fully mobilised a clamp is put across the pylorus and tissue forceps are placed so as to support the unclamped duodenum which is then cut across at the level of the ulcer Any tissue proximal to the ulcer makes closure of the duodenal stump difficult Every scrap of tissue beyond the ulcer is of value in effecting closure which is why it should not be clamped First the lumen of the duodenum is closed by a running catgut suture The suture line is then inverted by interrupted chromic gut or fine thread sutures in two or three rows The inversion should be free and easy Any reluctance on the part of the suture line to invaginate easily into the second part of the duodenum is unsatisfactory and either the sutures should be begun again (inversion may be facilitated by using a Connell type of suture for the initial duodenal closure) or more duodenum should be freed The latter step is fraught with danger from the risk of opening the common bile duct or more especially the main or accessory pancreatic duct It is the chronic ulcer penetrating the head of the pancreas which causes greatest difficulty especially when the dodenum is grossly shortened strictured or scarred

If access to the duodenum is at all difficult the pyloric end of the stomach can be divided between Payr s clamps The isolated antrum then forms a very convenient means of putting traction on the duodenum for its further dissection With the rare type of ulcer situated at the junction of the first and second parts or in the second part of the duodenum it is usually possible to divide and securely close the duodenum proximal to the ulcer Being bathed only in alkaline juices the ulcer soon heals During any of these procedures the gastro duodenal artery running down between the duodenum

and the head of the pancreas is frequently exposed and if injured may cause brisk bleeding but is readily controlled Closure of the difficult stump is discussed below

With the stomach freely mobilised the line of section is selected so that between two thirds and three quarters of the stomach is removed This often necessitates a higher separation of the greater omentum from the stomach A light holding clamp is applied at right angles to the long axis of the stomach and a little proximal to the proposed line of division Any vessels seen on the surfaces of the stomach close to this line are underrun and ligated The proximal jejunum is picked up where it emerges from beneath the transverse mesocolon An afferent loop of about eight inches but varying for individual patients is measured out and a light clamp applied to it engaging just sufficient of it to match the breadth of the stomach usually two to three inches Throughout this length the clamp should be evenly applied to the jejunal wall otherwise it may fail to control bleeding when the jejunum is incised and may possibly interfere with the nerve supply to the afferent loop (Burge 1956)

The afferent loop should be long enough to reach the divided stomach comfortably remembering that the stomach may retract for two or three inches beneath the costal margin when the clamps are removed The afferent loop may be taken to either curvature the writer takes it to the lesser which is the commoner practice The clamps on stomach and jejunum are now apposed and a two layer anastomosis carried out in the usual way The two ends of the anastomosis can be buttressed with a few interrupted stitches and the stomach is then allowed to retract beneath the costal margin The transverse colon and omentum are drawn to the right so as to leave the jejunal loops in relationship with the splenic flexure The afferent and efferent loops should lie comfortably and there should be no tension on the anastomosis Before the abdomen is closed the duodenal stump and head of pancreas should be inspected and a search made for any bleeding points especially near the spleen and in the great omentum If there is any doubt about the adequacy of the duodenal closure the tip of the Ryle s tube should be threaded through the anastomosis and placed in the afferent loop so as to keep it decompressed In addition a split tube rubber drain may be laid beside the duodenal stump and brought out through a stab incision

When the Polya gastrectomy is done for a gastric ulcer the closure of the duodenum is of course simple but the dissection of the stomach more difficult A chronic gastric ulcer attached to the pancreas can be separated and at the same time usually perforated by pinching it off that organ or by blunt dissection The base of the ulcer is left on the pancreas it should not be diathermised but can be gently swabbed clean and possibly dusted with penicillin or neomycin powder With a posterior gastric ulcer too the

identification and ligation of the left gastric vessels may be relatively difficult The case of the very high gastric ulcer on the lesser curve is considered later

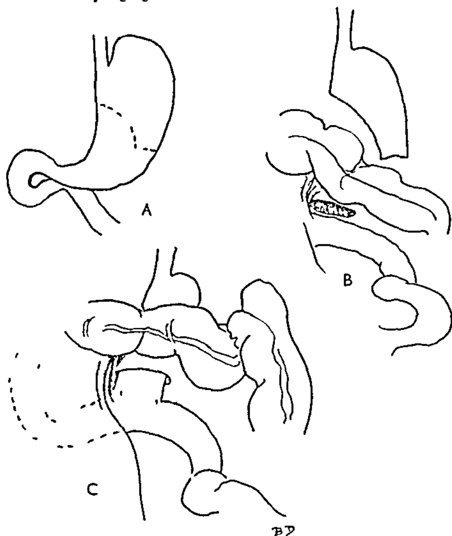


FIG 22
Hermon Taylor s partial gastrectomy A gastric tube is fashioned mainly from the fundus and greater curvature (A) and is then drawn down through an opening in the mesocolon beside the ligament of Treitz (B) to be anastomosed to the most proximal portion of the jejunum (C)

Retro colic gastrectomy

Hermon Taylor (1959) describes excision of the greater part of the lesser curve and ligation of all the vasa brevia with the formation of a gastric tube from the greater curvature This is led through the transverse mesocolon immediately beside the ligament of Treitz where it is anastomosed to the first loop of jejunum (Fig 22) This procedure avoids the formation of an afferent loop and excludes the formation of foramina through which herniation may

later occur Particularly good results are claimed in a series of 54 cases all observed for over 10 years

The management of the difficult duodenal stump

By the difficult duodenal stump is meant that most unpleasant and dangerous stump wherein lies an active inflamed and usually penetrating ulcer Most of the first part of the duodenum may be involved in a callous inflammatory mass Normal anatomy is distorted or obscured the tissues are friable and oedematous and it may be found either not possible to free sufficient healthy duodenum to ensure a sound closure or that adequate mobilisation and closure would endanger the main bile and pancreatic ducts It is of course always better to recognise and face the danger early and to change the plan of the operation one of the following procedures may be used either to avoid or to overcome the difficulty



FIG 23
Two stage gastrectomy The blood supply to the antrum must be intact (A) The antrum (B) must be removed within eight weeks otherwise recurrent ulceration is liable to develop rapidly

1 VAGOTOMY AND GASTRO-ENTEROSTOMY When the difficulty in the duodenum is appreciated before the stomach has been mobilised vagotomy and gastro enterostomy can be carried out instead of a Polya gastrectomy If the omental attachments of the stomach have already been divided the writer s modification of the Billroth I operation (*q v*) can be performed

2 TWO STAGE OPERATION This staged method of dealing with the difficult duodenum was described by McKittrick *et al* (1944) If the reaction around the duodenal ulcer is so extensive that it is obviously impossible to deal with it the stomach is divided three inches proximal to the pylorus these three inches must be left to allow for closure by inversion The divided

antrum is then closed deliberately leaving the pylorus and the duodenum untouched (Fig 23) The blood supply of the antrum from the right gastric and gastro-epiploic arteries must be preserved The gastro jejunal anastomosis is done in the usual way To prevent the development of a stomal ulcer (which *can occur very rapidly) the pyloric pouch must be completely removed within*

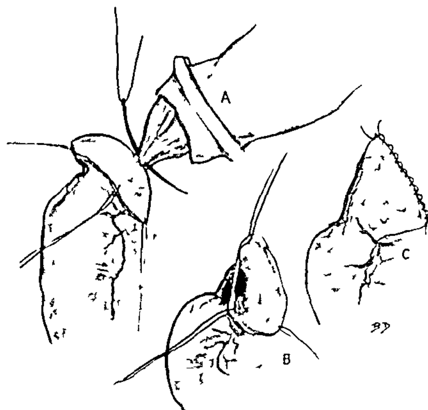


FIG 24
Bancroft s procedure of coring out and removing the mucosa of the antrum (A and B) and closing the seromuscular cuff which is left (C)

the next two months when much of the inflammation will have subsided This can usually be carried out easily and safely through a right subcostal incision with little disturbance The duodenal stump is then closed by inversion in the usual way

3 BANCROFT S PROCEDURE —*To avoid the need for a second operation* Bancroft (1932) recommended the removal of the hormone secreting mucosa of the pyloric antrum This is best done by having the assistant hold the stomach over *to the left putting the antrum on stretch The sero muscular coats of* the stomach are then incised in a circular fashion down to the mucosa from which they are dissected by reflection to the pylorus (Fig 24) Here the

mucosa is tied cut and dropped back The remaining sero-muscular coats are then closed by through and through sutures Inversion is impossible and the result is very untidy but quite satisfactory The blood supply of the antrum

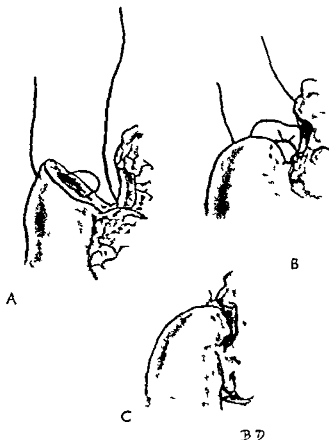


FIG 25
Nissen s manoeuvre The lateral cut edge of the duodenum is sutured to the infero lateral margin of the ulcer (A) A second row of sutures approximates the interior duodenal wall to the supero medial margin of the ulcer (B) thereby rolling the proximal end of the duodenum into the ulcer crater (C)

must be intact Qvist (1958) has recently described a method of fashioning the muscular cuff like a fish s mouth so that closure is easier and neater The sero-muscular layers of the pylorus are then formed into a cone the apex of which is invaginated with several purse string stitches

4 NISSEN S MANOEUVRE —The manoeuvre advocated by Nissen (1945) may be employed if the antrum has been devascularised before the difficulty is appreciated The ulcer is left undisturbed on the medial wall of the duodenum The free lateral edge of the divided duodenum is sutured to the lower edge of the ulcer using interrupted thread stitches (FIG 25)

The closure is completed by picking up the anterior duodenal wall half to three-quarters of an inch distal to the earlier line of sutures and stitching it to the free upper edge of the ulcer any available tags of omentum are tacked across Especial care must be taken to avoid damage to the gastro-duodenal artery A drain should be put down to this closure and the afferent loop drained by a naso-gastric tube

5 TEMPORARY DUODENOSTOMY —Welch (1949) and Priestley and Butler (1951) have recommended that if it is found impossible safely to close the duodenal stump a large rubber catheter (size (18 20F) should be tied into it and its end brought out through a stab incision under the right costal margin thereby creating a duodenal fistula (Fig 26) Very gentle suction is applied to this tube and intravenous fluid is given Often the discharge of bile and pancreatic juice lessen in quantity and cease within a few days and the tube can be removed This technique of temporary duodenostomy has recently been favourably reviewed by Lippert and Coleman (1958) Certainly it is safer purposely to create a duodenal fistula and begin energetic treatment immediately than to close the duodenal stump inadequately and so invite its rupture



FIG 26
Temporary catheter duodenostomy The catheter is secured in position by a purse string stitch

6 ANASTOMOSIS TO THE BILIARY TRACT The present writer (Wells and Brewer 1948) has pointed out that it is possible to anastomose the cut end of the divided duodenum to another viscus when it is impossible to close it by invagination Further the common bile duct may be near to involvement by the time the difficulty is apparent The duodenum can then be closed *and* by the same step an alternative pathway for the bile be established by end to-end anastomosis of the gall bladder (Fig 27) or common duct to the duodenum Hallenbeck (1958) has recently endorsed the value of this procedure which may also be used when penetration or fistula formation from a posterior ulcer has damaged the lower portion of the common duct

7 ANASTOMOSIS TO A ROUX Y LOOP —Under modern operating conditions no surgeon should ever leave the duodenum unsatisfactorily cared for All else failing a separate Roux Y loop of jejunum may be formed and

its proximal end taken up to the duodenum for end to-end anastomosis (Fig 28) When as a result of this arrangement the bile and pancreatic juice are directed away from the gastro jejunal stoma vagotomy is needed to give protection against jejunal ulceration

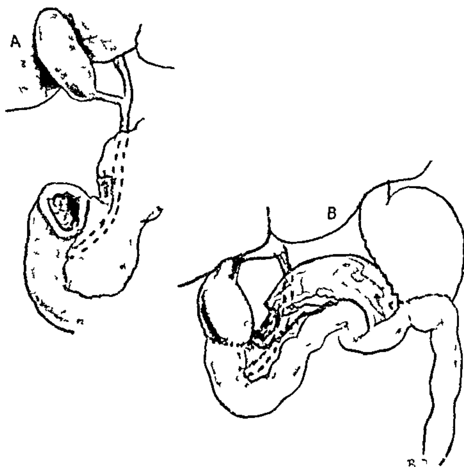


FIG 27
Anastomosis to the biliary tract not only deals with the open proximal end of the duodenum but provides an alternative pathway for the bile when the reaction around the ulcer is involving the common duct

Billroth I partial gastrectomy

SCHOEMAKER'S MODIFICATION The technique originally described by Billroth (1881 see Appendix) for gastric resection is no longer employed Billroth joined the open duodenum to the divided stomach near the lesser curve and separately closed the redundant open cut end of the stomach beside the greater curve Nowadays the anastomosis is made at the greater curve end of the divided stomach and the lesser curve is refashioned Most surgeons who want to re join the stomach to the duodenum after gastric resection use some variant of the technique described by Schoemaker (1921)

The stomach is freed as described for the Polya operation and the duodenum mobilised from the lateral aspect but less of it dissected out When the operation is being carried out for gastric ulceration the duodenum is divided just beyond the pylorus When a duodenal ulcer is present the first

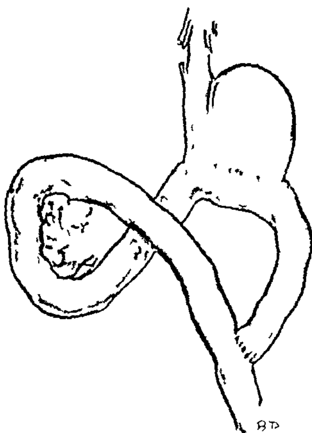


FIG 28
Anastomosis of the proximal end of the duodenum to a Roux Y loop of jejunum

part of the duodenum must be freed and the duodenum divided beyond the ulcer Care must be taken to avoid damage to the pancreatic and bile ducts As with the Polya gastrectomy the surgeon should decide at an early stage in the operation whether he is likely to be able to mobilise sufficient healthy duodenum for anastomosis to the gastric stump Gastro duodenal anastomosis is easier and safer than duodenal closure but if even for this purpose adequate mobilisation appears too difficult or hazardous the duodenum should be dealt with by either No 6 or No 7 of the emergency techniques described on page 111 and the operation completed by doing a gastro-jejunal anastomosis

Alternatively the writer's method of gastro-duodenal anastomosis (p 115) may be followed

Sometimes the whole of the cut end of the stomach can be *united to* the duodenum without reduction but it is more usual to reduce its width at the expense of the lesser curve Schoemaker (1921) used a special crushing clamp later modified by Morley (1939) when dividing the stomach but it is more usual to use ordinary Payr or other strong intestinal clamps The first clamp is put on at right angles to the greater curve grasping a width of stomach about one and a half times the width of the divided duodenum *It is placed at a point on the greater curve* which can be brought across to meet the divided duodenum without tension The stomach is divided between this clamp and another crushing clamp placed just distal to it across the part of the stomach to be removed A second rather larger Payr clamp is now applied along a line joining the tips of the first Payr clamp to the selected point on the lesser curvature of the stomach As it is easy to apply this clamp too high and so damage the oesophagus as it enters the stomach the oesophagus must be clearly demonstrated before the blades are finally pressed together The division of the stomach is then completed

The lesser curve is now reformed The blades of the longer Payr clamp *are oversewn with a continuous catgut stitch which is tightened as the clamp* is removed alternatively the free edge or crushed fringe of gastric tissue can be sutured This first suture line must then be invaginated either with a continuous stitch or with interrupted stitches If a continuous catgut stitch is used it can later be continued downwards to form the posterior outer layer of the gastro duodenal anastomosis and finally be brought up as the anterior outer layer thus completing the outline of the number '6 However the posterior serous layer can safely be omitted entirely in most cases one *careful* through and through layer being perfectly safe

The posterior all coats layer is made with interrupted sutures so as not *to restrict the duodenal lumen* If a serous stitch has not been placed behind it the interrupted sutures may be reinforced by oversewing with continuous catgut all coats without producing any constriction If sufficient care has been taken to avoid purse stringing the posterior layer the anterior layer can conveniently be made with a continuous Connell stitch Alternatively interrupted inverting sutures can be used or a Schmieden stitch but the latter produces rather a bulky line for inversion by the anterior serous layer and there*fore tends to constrict the stoma* There are obviously many possible variations of these techniques The general principles of end to-end suturing are discussed at the end of this section (p 120) Especial care must be taken to ensure a sound and water tight closure at the 'angle of sorrow the point where the reformed lesser curve meets the superior border of the *duodenum*

One or more triangular stitches should be put in taking good bites of anterior wall and then posterior wall of stomach and then of adjacent superior border of the duodenum These should not be tied too tight for fear of their cutting out

At the end of the operation the stoma should admit two fingers and should not be under any tension The anastomosis is gently lifted up and rotated so that its posterior surface can be seen and reinforced with one or more serous sutures if need be The subjacent head of pancreas can then be inspected Careful search must be made for any bleeding points and for leakage of pancreatic juice either where the duodenum has been dissected off the pancreas or where an ulcer has penetrated into it (Lavery and Kyle 1959)

WELLS MODIFICATION —The technique first described by the writer in 1957 is especially useful in cases in which the duodenum is greatly shortened and the ulcer adherent to the pancreas An abdominal vagotomy is done at the beginning of the operation The duodenum is mobilised together with the head of the pancreas in the usual way and is cut across just beyond the pyloric sphincter No attempt is made to separate the duodenum or its ulcer from the pancreas An incision is now made down the anterior surface of the duodenum from its open end for a distance of about two inches (Fig 29) If a Petz sewing machine is available the intended line of incision is crushed and fixed with clips to control bleeding Whether or not this has been done a long bladed haemostat is passed into the lumen of the duodenum and a coagulating diathermy current used to cut down on to it so as to produce the anterior duodenal incision one fifth of an inch away from the pancreas downwards from the open end for a distance of not more than two inches The duodenum cannot be clamped and any bleeding points that remain are either coagulated or underrun

A very limited gastrectomy is carried out the antrum only being removed The combination of hemigastrectomy and wide gastro-duodenal anastomosis may be followed in twenty per cent or more cases by intestinal hurry and post prandial symptoms in the writer s experience (unpublished data) Antrectomy alone with this anastomosis has a lower incidence of these complications

The cut end of the stomach is sutured to the duodenum first with a number of interrupted stitches through all coats to form a posterior line If the duodenum is deeply placed and difficult of access five or six of these sutures may be placed in position before any is tied The posterior suture line is completed by a continuous oversewing layer through all coats again The anterior suture line is made with a Connell stitch oversewn with a sero muscular Lembert suture These steps are identical with those just suggested in the suturing of the Billroth I anastomosis

Either the whole divided end of the stomach is attached to the duodenum or the part near the lesser curve is closed to reduce the size of the gastric opening as in Schoemaker s operation In the region of the ulcer the posterior and anterior suture lines may fall very close together and the stoma in this region may have little or no effective lumen but the resulting duodenal closure at the ulcer site is safe and sound

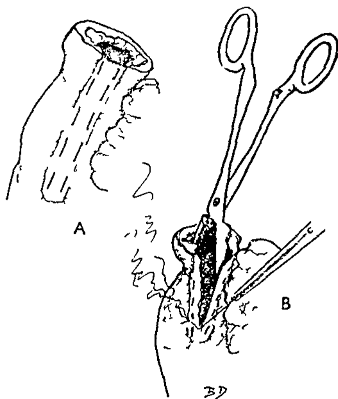


FIG 29
Anterior duodenal cut for the Billroth Wells type of gastro duodenal anastomosis Clips to control bleeding (A) may be inserted with a Petz clamp and the duodenal wall is divided with diathermy (B)

The advantages claimed for this method are that the risks inherent in the separation of a difficult ulcer are avoided and that a wide stoma is ensured The chief disadvantage of the operation is that it is impossible to avoid some soiling of the operation area with blood and duodenal content The latter is however bacteriologically sterile in cases of duodenal ulcer

Gastrectomy for high lesser curve ulcers

Gastrectomy for ulcers high on the lesser curve near the oesophagus may be carried out in one of three ways

1 A partial gastrectomy may be carried out below the level of the ulcer which is left *in situ* (Wells 1933) The rationale of this procedure is that

if the intragastric conditions after the operation are suitable for the prevention of recurrent ulceration they should also ensure the healing of the established ulcer The operation is of proved value and should be employed if operable malignancy can be excluded with certainty

2 The lesser curve may be divided without using clamps by repeatedly stitching and cutting from above downwards The first interrupted stitch is placed high up on the lesser curve above the ulcer and takes a bite of about one fifth of an inch of both walls of the stomach the stomach is then divided for a corresponding distance just below the stitch It is important not to place this first stitch too high up otherwise its later invagination may obstruct the opening of the oesophagus into the stomach The subsequent stitches and short incisions are placed around the ulcer and a little below and to the left of each other until the tips of the Payr clamp on the greater curve part of the stomach are reached The series of interrupted stitches which forms the new lesser curve is then invaginated

3 Total gastrectomy may be done if the lesion is suspected to be malig nant or in the event of repeatedly recurring anastomotic ulcer even when the antrum has been removed vagotomy performed and confirmed and non beta cell tumour of the pancreas and other hormonal causes excluded or dealt with

An operation of this character is more likely to develop unexpectedly in circumstances in which the operator has to improvise in accordance with accepted principles If the operation is planned a thoraco abdominal approach through the bed of the eighth rib and extending into the abdominal wall gives the best access The stomach is removed either by an extension of the technique already described or more radically in the case of suspected carcinoma (which is not relevant to the present volume) A Roux Y loop is prepared its proximal end closed and an end to side anastomosis made between the oesophagus and the jejunum

In making this anastomosis it is best having prepared the loop and closed its proximal end to select the site for the stoma At this point a one inch longitudinal incision is made in the antimesenteric side of the jejunum Next a number of through and through sutures are placed around the circumference of this opening to fix all coats of the small intestine together Three or five oesophago jejunal sutures are then carefully placed through all coats of the posterior layer of the oesophago jejunal anastomosis Not until all these sutures have been accurately placed are they actually tied When they have been tied any necessary additional sutures are put in The anterior suture line is then completed again with through and through inter rupted sutures tied on the mucosal surface

Secondary and more superficial sutures between the jejunal serosa and the surface of the oesophagus and fixing sutures between the jejunum and the parietal pleura are helpful and reassuring but will not compensate for any slackness in the placing of the through and through sutures which are the only ones that have any strong hold on the oesophagus and are those which appose the mucosal surfaces

Finally the diaphragm is repaired around the emerging oesophagus or jejunum and the chest closed after making provision for underwater drainage

Roux's gastrectomy-en Y

The stomach is prepared and the duodenum closed as for a Polya gastrectomy The duodeno-jejunal flexure is then identified and the upper jejunal vascular arcades inspected A site is chosen about two to three inches from the flexure where the vessels can be divided and a loop so prepared that it can be drawn up in front of the left part of the transverse colon or splenic flexure to reach with ease the level at which the stomach is to be divided When these steps have been planned the peritoneum over the jejunal arcades is incised to expose the vessels which are then divided between ligatures and the jejunum cut across between clamps

The end of the jejunum on the distal side of the cut is then securely closed and the loop thus prepared is taken up to the stomach

If vagotomy has already been done or is to be done an end-to-side gastro jejunal anastomosis is made at any convenient level on the stomach bearing in mind that the patient's future comfort is roughly proportional to the amount of stomach left behind If vagotomy has not been done and is not intended a near total gastrectomy is called for to keep down the risk of marginal ulceration (Schofield and Anderson 1953) The writer feels that this is not a very reliable safeguard and that the sacrifice of so much stomach is better avoided

The fork of the Y is now made by joining the proximal jejunum end-to-side with the distal loop about eight inches below the gastro-jejunostomy It must be remembered that the distal loop carries the main stream of undigested food from the stomach within minutes of its being swallowed It is important therefore that the fork of the Y should be made with especial care to avoid constricting this food channel A two-layer edge-to-edge technique serves this purpose well

The proximal end is drawn under the distal loop so as to lie to its left Then the parts being suitably clamped a one inch longitudinal incision down to but not into the mucosa is made on the antimesenteric border of the distal loop and a similar cut on the appropriate side of the proximal end

The sero muscular coats thus prepared are sutured to one another with fine continuous catgut and the end left long The lumen of the bowel on each side is then opened and a second running mucosal stitch placed in the posterior layer so that it *just* picks up the sero-muscular edge Turning the corner this suture is brought back as a Connell stitch along the anterior mucosal layer again *just* encroaching on the cut sero muscular edge The ends are tied and cut Then the anterior sero muscular edges are joined by bringing the original posterior sero-muscular suture round to the front and finally its ends also are tied off and cut

This technique is a little slow and looks somewhat untidy in its execution but the way in which it leaves the main lumen unrestricted is very striking Nissen (1958 personal communication) uses a somewhat similar one layer technique from which the mucosal suture is omitted entirely The sero-muscular layers are freed by making a cut down to the submucous layer and are then accurately and carefully sutured the loose edges of the mucosal cuff are left lying in contact with one another but unsutured

When the Roux-en Y operation is used in the presence of hiatus hernia (p 99) it is particularly important to make sure that the outlet from the gastro jejunostomy is also free and unencumbered since the food needs literally to *fall* out of the stomach into the jejunum there being no gastro oesophageal closure and therefore no *vis a tergo* It is tempting to take the jejunum up to the stomach through a hole in the transverse mesocolon but this attractively tidy arrangement has led to trouble as a result of the bowel becoming obstructed from without by a mass of fat necrosis at this site The ante-colic arrangement is preferred

Conversion from Polya to Roux Y (*e g* in afferent loop syndrome) is extremely simple Vagotomy is confirmed or performed and hiatus hernia looked for The afferent loop is then divided where it joins the stomach at the lesser curve and closed at its distal (stomal) end The proximal end is then drawn under the efferent loop to its left side and there joined as just described

At the conclusion of these procedures the window resulting from the mesenteric incision is carefully closed to lessen the risk of internal herniation It is not recommended that any attempt should be made to seal the wide opening which exists between the efferent loop and the transverse colon as has been suggested by Stammers (1954) in the performing of a routine Polya gastrectomy

Colonic and jejunal transplants

Jejunum may be transplanted isoperistaltically in order to bridge a gap *e g* between the oesophagus and the duodenum in a total gastrectomy Colon may be transplanted to bridge a gap to serve as a reservoir or to delay the

onward movement of the gastric contents by its antiperistaltic action The only procedure that can usefully be described in any way formally is that of Moroney (1951) If the operation is foreseen a full bowel preparation is prescribed

The stomach and duodenum are separated and prepared for end to-end anastomoses A convenient part of the transverse colon is then selected and its vessels inspected with a view to isolating a segment of bowel about four to six inches long with its blood supply intact End to-end continuity of the colon is then restored and the isolated segment, without rotation joined end to-end with the stomach and duodenum so that its peristalsis runs against the food stream Interrupted sutures are used for all the through and through layers Vagotomy is essential to prevent recurrent ulceration

Isoperistaltic jejunal interposition between the oesophagus and the stomach prevents gastro oesophageal reflux and bilious regurgitation and should be considered with vagotomy in cases of cardio-oesophageal incompetence Patients who have had this operation may be unable to vomit in any circumstances

General remarks on technique

CONTRACTED BOWEL —Under modern anaesthesia using relaxants and minimal quantities of narcotic agents the intestine is not paralysed as it is and regularly was under ether and chloroform It therefore responds to handling by vigorous contraction which causes both thickening and shortening The thickening materially increases the technical difficulties of anastomosis and the shortening leads to gross errors of judgment of intestinal lengths It may be taken as axiomatic that surgeons regularly underestimate the length of selected loops which become longer by a factor of possibly three or four when the viscera regain their normal tone These facts must be borne in mind

END-TO-END ANASTOMOSIS —In making intestinal anastomoses the tendency is more and more to favour end to-end techniques even when there is disparity between the diameters of the parts to be joined One desirable effect of end to-end anastomosis is the avoidance of blind ends which are inevitable in side by side techniques Any blind end into which peristalsis tends to project the intestinal content is liable to become loaded and distended even if it is only a few inches long The deceptive shortening of contracted intestine in modern anaesthesia increases therefore the desirability of end to-side techniques since the blind ends left behind may prove to be much longer than was thought at the time of operation

In making intestinal anastomoses the need for and even the desirability of multiple layers of sutures has been called in question and there is a trend towards simplification This is most clearly seen in the surgery of the oesophagus and of the large intestine which barely come into the scope of this present work In these latter situations maximum emphasis is laid upon the all-coats suture in the case of the oesophagus because there is no peritoneal covering and the more superficial sutures have little hold and in the case of the colon because accurate apposition of the mucosal coats is the prime need Provided the surgeon is satisfied that his all-coats layer is sound he is at pains not to endanger it by placing around it further sutures which may damage the blood supply or cause undesirable pocketing or constriction In the making of these vital all-coats end to-end anastomoses interrupted sutures minimise the risk of strangulation of tissue and can be accurately placed and obviate the reduction of the lumen by any purse string effect That this effect is particularly important today will be evident when the already contracted state of the viscera in modern anaesthesia mentioned above is taken into account These principles can be usefully remembered on all occasions in all gastro intestinal surgery

Ligature and suture materials —The choice of material for ligatures and sutures is a matter for the individual surgeon but one or two points are worthy of consideration The non absorbable materials—linen thread cotton silk and synthetic materials—probably produce little immediate reaction in the tissues and are very well tolerated in the absence of infection In the presence of infection their foreign body effect is certainly a disadvantage and organisms within the interstices of twisted multiple fibres may be inaccessible to the antibiotics Continuous thread or similar sutures are to be avoided for a variety of reasons When they are circumferential they limit permanently the size of any lumen they surround outside the lumen of the gut an abscess may be formed inside some part of the suture may ulcerate into the lumen of the viscus and remain hanging therein through the mucous membrane the bowel wall being thereafter subject to recurring infection In the urinary tract calculi form on such tags of thread and experience in their removal proves that the thread remains firmly in the tissues even for years contrary to the common belief that in such circumstances it comes away fairly quickly

The advantages of thread (and similar materials) are its permanence its tensile strength and its good grip against slipping either on the tissue or on itself

Catgut is very much more expensive than the other materials but is extremely reliable and uniform today It is difficult to think of any situation in which a suitable grade of catgut is less reliable than a non absorbable

suture and its eventual disintegration and absorption allows much greater freedom in the selection of lengths and strengths

Finally it may be said that non absorbable *materials should be* as delicate as will suffice for their task and that they should be used sparingly and only as individual knots or interrupted sutures

INTUBATION —It is routine practice today to introduce a Ryle s or similar tube into the stomach before the patient goes to the theatre and to retain it during some part of the convalescence *Only when ileus or obstruction* develop is any more ambitious programme of intubation essayed Then when the patient s condition may make the manoeuvre very distressing attempts *may be made to introduce a Miller Abott* Cantor or other tube into the jejunum to retrieve the situation The desirability of having a tube in the afferent loop whenever there is any doubt about the soundness of closure of the duodenum has already been emphasised but few surgeons have the neces sary variety of tubes readily available or are practised in the rather tedious art of introducing them at the time of operation with the assistance of their anaesthetist into appropriate segments of the intestine

During the last three years the writer has regularly employed in all cases of major abdominal surgery a long polythene tube introduced by the anaes thetist to the stomach and threaded onwards by the surgeon into the jejunum Whilst much work on this subject is being done and remains to be done *it is significant that the anxious problems of post operative ileus* and obstruc tion have virtually disappeared even in such previously worrying cases as total cystectomy with transplantation of ureters pelvic evisceration and so forth

Intragastric tubes are aspirated intermittently but if any large quantity of gas or fluid is coming up continuous suction should be used on these and on intra intestinal tubes The best method of setting up such suction is by a system of Wangensteen s bottles which is thoroughly reliable and never builds up sufficient negative pressure to block the tubes by sucking the mucous membrane into their eyes

Intra jejunal tubes can be utilised for early feeding before fluids can be accepted by mouth Experience suggests that milk should be avoided as being likely to initiate or encourage diarrhoea whether of bacterial allergic chemical or other origin

DRAINAGE AND DRAINS —If caution demands the *placing of a drain* e g down to the duodenal stump it is at least worthwhile putting in a drain which will drain For this purpose a soft rubber tube split in its length from end to end is excellent It has an open channel to *which there is access from* end to end through the slit It can be rotated pulled out and pushed in

Corrugated rubber used flat is of little use Its corrugations rapidly become filled with fat granulation tissue and the like If a roll of corrugated rubber is used it amounts to a tube but is less easily managed Penrose drain is useful in the pelvis for example when there is a cavity to be filled but has little to be said for it in the upper abdomen

The management of a biliary duodenal or pancreatic fistula calls for sump-suction drainage (p 130) For this purpose a wide tube is passed into the wound and a second smaller tube is introduced down its lumen almost but not quite to the bottom Between the tubes there must be room enough for a free current of air Suction is then applied to the smaller tube so as to carry away any fluid as it accumulates in the sump at the bottom of the larger tube

Post-operative management

At the end of the operation the Ryle s tube is aspirated in order to be sure there is no excessive bleeding into the stomach If there is too much fresh blood as may occasionally happen the stomach is reopened and the suture lines oversewn

The gastric tube is aspirated at hourly intervals and when bowel sounds return an ounce of water hourly is given by mouth Although it may take much longer the quantity aspirated from the stomach often becomes less than the amount taken as drinks in from two to four days The gastric tube can then be removed It should be re introduced at once if there is the slightest doubt about the satisfactory emptying of the stomach This may be manifested by a reluctance to take more drinks the occurrence of vomiting or hiccup or the onset of signs of acute dilatation of the stomach namely circulatory collapse increased respiratory rate epigastric discomfort and particularly restlessness and insomnia In such a case the emptying of the stomach with Wangensteen s bottle apparatus gives such immediate relief that the patient commonly goes to sleep whilst the process is going on

After a Polya gastrectomy the appearance of bile in the aspirate is an encouraging sign that the afferent loop is draining well into the stomach The non appearance of bile especially if associated with unremitting upper abdominal discomfort may be a valuable warning of increasing tension in a poorly draining afferent loop and of the likelihood of a duodenal blow-out

If a jejunal tube is in use it should be retained until the patient is am bulant taking a reasonable diet and having a normal bowel action This is often unwelcome to the patient who resents the undoubted discomfort of the retained tube Its replacement is however impossible once it has been taken away and its value as a prophylactic measure and sometimes for feeding is

very considerable and well worth while Ordinarily no fluid is recovered from this tube which is however left open and appears to function by decompression of the intestinal lumen

All the fluid that is aspirated is saved measured added to urine and any other secretion or discharge and the whole sampled for its electrolyte content In this way it is possible to gauge the fluid and electrolyte replacement needed from day to day without the necessity of frequent blood analyses electrocardiographic records and so forth

Appropriate intravenous therapy is instituted immediately after the operation and continued until oral or jejunal feeding is possible Whilst oral take over may be achieved in under forty eight hours it may sometimes be delayed for as long as a week especially after vagotomy and seemingly not infrequently after gastro duodenal anastomosis It is in such cases that the jejunal tube is especially useful for feeding Welbourn (1956) has reported non emptying of the stomach for twenty six days in a patient in whom no sign of obstruction was found when the abdomen was re-opened and whose ultimate recovery was completely satisfactory Delays of much the same order have been within the writer s experience

The details of fluid and electrolyte management are not peculiar to gastric surgery and will not be discussed further

Deep breathing exercises are instituted before the operation and continued immediately afterwards under supervision by the physiotherapist She also massages the lower limbs and is responsible both for passive exercises and for seeing that the patient carries out active exercises especially of the lower limbs at intervals throughout the day Ambulation which begins on the first post operative day is no substitute for these exercises which serve to lower to vanishing point the incidence of post operative thrombosis and embolism

When fluids can be taken freely the intake is gradually increased so that in a favourable case a light but varied and reasonably normal diet is being taken at the end of ten days Meals should be small and frequent and this rule should be adhered to for several months On the other hand an 'ulcer diet is specifically *not* recommended The gastrectomised patient in particular is in need of a good intake of foodstuffs rich in protein calories iron and vitamins An iron supplement in the diet (or even intravenously) is often advisable and especially so in women before the menopause The patient must be followed up at regular intervals for some years after his discharge from hospital so as to prevent or deal promptly with any late complications or deficiencies which may develop

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ADDENDA

1 It has been widely held to Finsterer s discredit that he failed to appreciate the part played by the antral mucosa in peptic ulceration Kinsella (1959) however draws attention to the fact that it was he who in 1918 first stressed the need to ablate the lining of the retained antrum in order to control the hormonal secretion of acid in accordance with Edkin s hypothesis

2 *Antrectomy* Recent observations suggest that the output of gastrin may continue after removal of the antrum if the resection is not continued to a relatively high level up the lesser curve

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CHAPTER VII

THE EARLY COMPLICATIONS OF OPERATIONS ON THE STOMACH

By James Kyle and Ian W MacPhee

THE complications of gastro enterostomy and partial gastrectomy fall into three groups namely those specific to the operations themselves those resulting from abdominal section and the general complications which may follow any surgical operation Apart from haemorrhage and shock the second and last groups do not require full consideration here and attention is directed mainly to those complications which are due to the operative procedures on the stomach and duodenum All are serious but they are fortunately uncommon Those most likely to be encountered are haemorrhage rupture of the duodenal stump and pancreatitis

Haemorrhage

Bleeding after gastric resection occurs in about one per cent of patients one in ten of patients so affected dies (Wilkins *et al* 1957) The bleeding usually takes place immediately and during the first day or two after gastrectomy It comes mostly from the suture line and only rarely from omental or other vessels which have escaped detection at the time of the operation or from an ulcer which has been left behind The amount of blood lost is frequently small but occasionally large quantities of blood and clots are vomited or aspirated In the latter event the patient fails to make a proper recovery following the operation and all the signs of continued bleeding are present namely pallor rapid feeble pulse hypotension dyspnoea restlessness sweating and possibly loss of consciousness Where the bleeding has occurred from for example omental vessels or perhaps from the hilum of the spleen the general signs of haemorrhage and of peritoneal irritation are found Non obstructive jaundice may later result from the absorption of pigment

Minor degrees of bleeding after gastrectomy mostly cease spontaneously when treated by sedation and a slow blood transfusion Haemorrhage of a degree sufficient to induce serious signs must however be treated energetically Immediate and rapid blood transfusion is necessary and the stomach is aspirated and washed out Unless bleeding quickly ceases laparotomy should be performed without delay The abdomen is re opened and a rapid inspection carried out for a bleeding vessel or a large haematoma in the mesentery or behind the peritoneum If no appreciable bleeding has taken place into

the peritoneal cavity clamps should be applied on both sides of the stomal suture line and the anterior sutures removed Blood should be swabbed from the mucosa and in turn each clamp relaxed and the cut edges inspected Large vessels should be separately underrun the posterior suture line over sewn and the anterior line remade The interior of the gastric remnant should be inspected in case a high ulcer has been overlooked Some surgeons prefer to open the stomach above the anastomosis and to deal with bleeding vessels and oversew the suture line from within During all this time blood trans fusion is continued and the patient should be recovering from the effects of bleeding before leaving the theatre for the second time

In a few cases there may be secondary and usually slight bleeding about the tenth post-operative day possibly caused by sloughing at the suture line Littler (1957) has recently drawn attention to the post gastrectomy combina tion of pain fever and severe secondary haemorrhage the bleeding occurs from ten to twenty days after operation and is believed to be due to sepsis at or outside the anastomosis

Re activation of a retained ulcer or the development of a stomal ulcer should be suspected if bleeding first appears more than three weeks after operation

Shock

Since most examples of operative shock are due to blood loss the subject of shock may appropriately be considered at this point but shock present from the time of operation is now a rare complication of gastrectomy When analysing the mortality in a series of about 5 000 gastric operations Marshall and Reinstine (1957) found that nine per cent of the 254 deaths were attributed to shock the frequency of this diagnosis however decreased considerably when autopsies were performed as these examinations usually revealed some organic cause of death Hypovolaemic shock due to a diminution in the blood volume is usually the result of blood loss during the operation such loss may be greater than the surgeon realises at the time Occasionally a patient with a normal blood picture before operation may because of prolonged under nourishment be suffering from a reduced blood volume which becomes apparent only when post-operative hypovolaemic shock develops as a result of further blood loss and fluid deprivation This combina tion of circumstances is even more likely to lead to anxiety during anaesthesia especially with controlled respiration in which the absence of negative intra thoracic pressure results in a diminished return of blood to the heart which in turn aggravates the previously unrecognised hypovolaemic state Most of the sedative drugs used routinely after operation depress respiration and

consequently, in the same way diminish the return of blood to the heart The resultant clinical picture again closely resembles and is indeed a special form of shock

Errors of anaesthetic technique or excessive traction on the viscera during light anaesthesia can cause a serious fall in blood pressure which may be difficult to distinguish from true hypovolaemic shock In a few patients unsuspected adrenal insufficiency may account for an otherwise unexplained fall in blood pressure during and after anaesthesia Adrenal atrophy is a source of danger in patients who have been given cortisone even though this may have been discontinued for many months

Hypovolaemic shock is sometimes confused with coronary thrombosis or acute left ventricular failure The recognition of pulse irregularities venous engorgement respiratory difficulties and the finding of characteristic E C G changes should indicate the true diagnosis It should be noted that when gastric surgery is performed in patients with pre-existing heart disease a mortality rate of nearly thirty per cent has been recorded (Marshall and Reinstine 1957)

Massive collapse of the lung can cause a clinical picture similar to operative shock but it usually occurs after the first twenty four hours and examination of the chest reveals dullness and loss of air entry on and a mediastinal shift towards the side of the collapse

In short shock after gastric surgery should always be regarded as a clinical state for which an adequate cause can be found in the great majority of cases True primary shock is a diagnosis that is seldom justifiable

True hypovolaemic shock is treated initially by restoring the blood volume to normal sufficient blood is transfused to compensate for all losses If transfusion fails to raise the blood pressure l noradrenaline (Levophed Bayer) 4 ml in 500 ml of five per cent dextrose solution may be given slowly There is some risk of tissue necrosis with this substance (Humphreys et al 1956) especially if a cut down drip is in use A catheter in a large vein permits greater dilution of the drug as it runs in and eliminates this risk Alternatively 10 mg of either methoxamine B P or phenylephrine B P in 250 ml of five per cent dextrose solution intravenously may be preferred Persistent hypotension and failure to react to these measures suggests un recognised adrenocortical insufficiency and intravenous hydrocortisone 100 mg in 500 ml five per cent dextrose should be given empirically (Dundee 1957) Cortisone must be prescribed as a routine in any patient who has received this hormone for no matter what condition during the preceding two years

Leakage from the duodenal stump, or from the anastomosis

These complications occur within a week of the operation and may prove fatal The leakage may result in a small localised abscess a rapidly enlarging and easily recognisable fluid collection or in a disseminated and often fatal peritonitis A blown duodenal stump is much commoner than a leaking anastomosis and usually follows a Polya operation for a penetrating duodenal ulcer Peritonitis accounted for thirty seven per cent of the deaths of patients with duodenal and stomal ulcers operated on at the Lahey Clinic a blown duodenal stump was responsible for the peritonitis in more than half of the fatal cases (Marshall and Reinstine 1957)

Leakage from the duodenal stump may be precipitated by any one of the following factors

1 Obstruction to the afferent loop

2 Inadequate closure of an extensively scarred distorted and oedematous duodenum

3 Devascularisation of the duodenal stump when preparing the cuff before closure and inversion

4 Local pancreatitis

Almost invariably the first factor combined with one of the others is the ultimate cause of rupture of the duodenal closure It should be noted that acute distension of the afferent loop without any leakage may prove fatal (Watson 1958)

If at the time of the operation the stomach tube can be directed across the anastomosis into the afferent limb and if continuous suction is applied to keep the loop empty the danger of leakage is reduced considerably A Y shaped tube can also be used for suction the longer limb is passed into the afferent loop whilst the shorter remains in the stomach Should the actual closure of the duodenal stump be inadequate a large bore drainage tube can be led down to the site of the closure This may be left in place for some days and if leakage does occur a localised abscess and direct fistula may result and a large extending abscess or diffuse peritonitis be prevented On the other hand it is at least possible that the tube itself may be a factor in the causation of a duodenal leak and routine drainage in a Polya operation cannot be regarded as good practice Rarely and when adequate closure is quite impossible a catheter may be sutured into the open end of the duodenum and a duodenal fistula accepted *ab initio* (see also p 111)

Obstruction of the afferent loop should be suspected if bile fails to appear in the gastric aspirations within the first day or two after a Polya type of operation Later the patient may complain of vague pain in

the right side of the abdomen and occasionally an ill-defined mass is palpable The patient's general condition *does not show that rapid improvement* usually noticed within a day or two after the operation The temperature rises slightly the pulse is elevated the patient may look anxious and there may be general abdominal distension due to ileus Straight X ray examination of the abdomen is sometimes helpful as the dilated afferent loop may be visualised In such circumstances laparotomy is advisable to relieve the obstruction

If after prophylactic drainage duodenal leakage does occur a small bore double channel catheter (Fig 30) or tube (Fig 31) should be passed bore double channel catheter (Fig 30) or tube (Fig 31) should be passed into the fistula and continuous 'sump' *suction applied The secretions thus collected* are filtered and returned to the stomach by the Ryle's tube The skin must be protected with an aluminium paste or silicone preparation or swabs soaked in skimmed milk

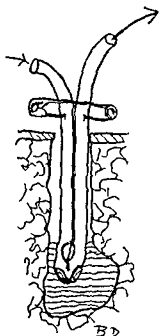


FIG 30
Sump suction using a doubled up catheter Lateral openings should only be cut in the catheter where it bends

In the absence of preliminary drainage a duodenal blow-out is a major catastrophe which is most likely to occur between the second and the seventh post-operative day—classically on about the fourth day There is sudden pain and collapse which may be *mistaken for a massive coronary or pulmonary infarct* When a duodenal blow-out is diagnosed adequate drainage must be instituted at once and when combined with supportive therapy is life saving in about half the cases A major laparotomy should be avoided An incision of two or three inches is made below the right costal margin at or lateral to the nipple line a tube *guided by the finger passed down to the duodenal* stump and sump suction drainage instituted

A fatal outcome in these cases may be due to various causes of which some are avoidable Digestion of the abdominal wall if not controlled by sump-suction may lead to extensive disruption of the main incision which in turn it may be impossible to repair Fluid protein and electrolyte loss become serious *if there is difficulty in returning the secretions through the* Ryle's tube *via* the stomach into the jejunum intravenous fluid and electrolytes are always required A *jejunostomy for feeding* made through the *smallest possible incision* well to the left of the abdomen is sometimes invaluable especially if methylene blue taken by mouth appears rapidly out through the fistula An indwelling jejunal tube introduced at the primary

operation is often useful for feeding purposes Finally especially if the emergency operation has been delayed generalised peritonitis may not be avoided and in these circumstances is almost certain to prove fatal

In all cases where leakage from the duodenal stump is expected or is established full doses of penicillin and streptomycin should be given parenterally When a duodenal fistula has developed propantheline bromide (Probanthine Searle) in doses of 15 mg four times daily reduces the pancreatic and gastric secretion and is of value in diminishing the amount of leakage and in assisting the closing of the fistula Nursing the patient on his left side with the head of the bed raised also helps it is claimed to diminish the volume of juice escaping through the fistula (Chambler 1958) With conservative management a duodenal fistula usually closes spontaneously in about three weeks unless the afferent loop is obstructed in which case an operation to free it is necessary though seldom possible

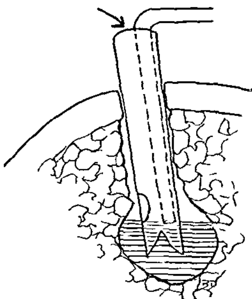


FIG 31
Sump suction using a large rubber tube Suction is applied to the small glass tube passing down inside it

Leakage from the site of the anastomosis may occur after the Billroth I (Fig 32) or Polya type of operation In neither case is the condition common and usually it amounts to no more than a small peri gastric abscess Occasionally however a large part of the suture line may burst or the stomach adjacent to the closure may have been devascularised and considerable soiling of the peritoneal cavity may result In such circumstances profound collapse occurs and the outlook is grave In all cases in which it seems likely that leakage has taken place it is safer to re-open the abdomen at once than to temporise This is especially true if gastric aspiration has been unsatisfactory and has not been keeping the gastric remnant empty Radiological techniques to demonstrate the leak such as injecting contrast medium down the nasogastric tube are unreliable and air from the original laparotomy will still be present below the diaphragm A defective Polya anastomosis can usually be refashioned or repaired but a leaking Billroth I anastomosis may have to be converted into a Polya using a temporary catheter duodenostomy to deal with the duodenal stump Vigorous resuscitative measures are necessary

Occasionally if early operation has not been resorted to it may be necessary in a few days time to drain a large subphrenic or right sided or even pelvic collection of fluid or later pus



FIG 32

The angle of sorrow Barium meal showing a left subphrenic abscess arising from a leaking Billroth I gastro duodenal anastomosis Recovery followed drainage of the abscess

Pancreatitis and pancreatic leakage

Fatal pancreatitis occurs in about one per cent of patients submitted to gastrectomy according to Burton *et al* (1957) lesser degrees of pancreatitis may be rather more common and may account for some of the previously unexplained cases of peritonitis and duodenal rupture One of the following operative errors usually unrecognised at the time may be responsible for the condition

1 The unrecognised presence of the pancreatic (and biliary) duct *in* the exposed floor of a penetrating duodenal ulcer

2 Division or ligation of the accessory pancreatic duct especially when it is the principal outlet for the pancreatic secretion (Millbourn 1949) Injuries to the main duct are much rarer but the scarring from a duodenal ulcer can draw the ampullary region proximally into a dangerous position

3 Excessive handling of the pancreas during the mobilisation of a difficult duodenum

4 The inadvertent cutting away of a piece of pancreatic tissue when dissecting an ulcer off the pancreas

5 Interference with the blood supply to the pancreas particularly when the arterial arrangement is anomalous (Burton *et al* 1957)

Far and away the most important of these accidents is the inadvertent division of an important pancreatic duct This may arise in the dissection of the shortened duodenum resulting from chronic ulceration It is commoner after the more extensive duodenal mobilisation necessary in an operation of the Polya type Constant watch must be kept for the accessory duct in order to avoid damaging it Any strands of tissue in the concavity of the duodenum which have to be divided should be carefully tied with unabsorbable thread A finger in the duodenum may feel the papilla at the orifice of the ampulla of Vater but can rarely locate the tiny proximal papilla of the accessory duct At the end of the operation the head of the pancreas should be carefully inspected to see if any clear pancreatic juice has escaped Wallensten (1958) however believes that stagnation within the duodenum or afferent loop of a Polya gastrectomy may be the cause of the condition

Post-operative pancreatitis or peritonitis due to leakage gives rise to pain at such an early stage that it is difficult to distinguish it at first from the post-operative discomfort of which many patients complain The possibility of this rare complication should always be considered when a reasonable patient fails to get relief from pain with an adequate sedative There may be some elevation of the pulse rate and temperature and the pain becomes increasingly severe The serum amylase level is usually but not always markedly raised

Pancreatitis and leakage require prompt and vigorous treatment Adequate drainage is provided for any escaping secretions and propantheline bromide (Probanthine Searle) 15 mg four times per day is administered in order to decrease the volume of pancreatic secretion Antibiotics are mostly given but are unlikely to have any significant effect on a predominantly chemical peritonitis Gastric aspiration and intravenous fluids are continued until the condition has settled

Delayed emptying of the gastric remnant

In the early post-operative period persistent vomiting is most likely to be due to delay in emptying of the gastric remnant intestinal ileus or mechanical obstruction Some delay in the evacuation of stomach contents is usual after most gastric operations and is controlled by aspiration Under this regime the condition almost always clears up within a matter of days Occasionally failure of the gastric remnant to empty promptly persists for

longer and may lead to dehydration and electrolytic deficiency This complication seems to be commoner in America than in Britain It is said to be due to oedema at the anastomosis although no evidence has been presented to prove this Hypoproteinaemia is sometimes responsible for slow emptying and potassium deficiency is another possible cause (Logan 1953) Radiological examination generally shows an increased resting content of gastric juice and delay in emptying Only rarely can a true organic stricture or narrowing be seen but Wells (1958 personal communication) has found complete obstruction due to fat necrosis in a number of cases

This stomal obstruction seems most likely to arise in cases in which the abdomen has been opened on a previous occasion Probably interference with omental or mesenteric blood supply leads to localised fat necrosis The necrosed area becomes densely adherent to the stoma from which it cannot be separated and so if fat necrosis is discovered a new anastomosis must be made so as to short-circuit the obstruction the close investment of the stoma defies dissection An indwelling jejunal tube for either aspiration or feeding is a helpful adjunct in all cases Occasionally no obstruction can be found Welbourn (1956) has reported such a case in which delay in emptying persisted for twenty four days without discoverable cause even at laparotomy

Rarely in the immediate post-operative period the gastric remnant may completely fail to empty causing (in the absence of effective aspiration) acute dilation of the stomach

Slow emptying is a recognised and frequent sequel of vagotomy even though a gastro-enterostomy or partial gastrectomy has been carried out as well Some delay in emptying is almost invariable for a few weeks after the Billroth I type of operation but the delay is not associated with symptoms and radiological investigations at six weeks and three months after gastrectomy show normal emptying

Treatment is directed towards keeping the gastric stump empty by means of continuous suction and correcting fluid and electrolyte loss by intravenous therapy If symptoms persist for many days and especially if there is pain then some form of organic obstruction at or close to the stoma must be suspected and re operation is necessary

Vicious circle vomiting

This complication namely the regurgitant and copious vomiting of bile stained gastric contents occurs after gastro-enterostomy but is now rarely encountered The condition develops in the early post-operative period and may be considered in some respects similar to delayed emptying of the stomach after gastrectomy It has always been suggested that the mechanism

is a return of gastric and duodenal contents to the stomach *via* the afferent loop of the gastro-enterostomy When gastro-enterostomy was an everyday operation a well positioned stoma seldom gave trouble The placing of the stoma in the pre pyloric region of the stomach was held to be responsible for vicious circle vomiting in most cases Fat necrosis around the stoma is another possible cause

Treatment at first is by continuous suction drainage but should this prove ineffectual it may be necessary to re operate and possibly to convert the gastro enterostomy to a partial gastrectomy If this is necessary it should be done before the patient has become too dehydrated Recent surgical literature is singularly devoid of references to this complication

Paralytic ileus

Paralytic ileus is not a common complication of partial gastrectomy It may closely simulate mechanical obstruction of the small intestine and in the early stages it may be difficult to distinguish between the two conditions In ileus large quantities of gastric and intestinal juices are aspirated there is effortless vomiting and on auscultation no active bowel sounds are heard Distension is marked A prophylactic jejunal tube (p 122) is invaluable in preventing ileus and in dealing with obstruction if it occurs

With established ileus the aims of treatment are firstly to keep the stomach and small bowel empty by aspiration through a jejunal tube or the more usual type of gastric tube and secondly to replace the large quantities of fluid and electrolytes that may be lost from the body The computation of the fluid and electrolyte requirements is best done by the simple process of adding together the quantities lost by different routes It is necessary to save every specimen of gastric aspirate vomitus urine faeces and the discharge from any drain or fistula All the specimens from a twelve or twenty four hour period are mixed together (a homogeniser being useful for this purpose) the total volume is noted and the electrolyte content determined The amounts lost are then made good by suitable intravenous fluids alcohol may be added to provide additional calories Blood and plasma are valuable especially if the plasma proteins are low Amino acids are given intravenously when necessary but tend to cause venous thrombosis which is highly inconvenient in exactly this type of case in which prolonged parenteral therapy may be called for The passage of a small bore polythene tube *via* a peripheral vein into the vena cava is a convenient way of ensuring patency and facilitates the administration of more irritant substances such as amino acids which are poorly tolerated by the small veins

If in spite of everything and in the absence of an indwelling jejunal tube the ileus persists the passage of a Miller Abbott tube may be attempted

However most surgeons lack experience in the use of this instrument and few have had success with it It is probably wiser if the patient is fit enough to re open the abdomen and seek any possible cause of the trouble A jejunal tube can then be passed *per vias naturales* Alternatively if the patient is too ill for a major exploration a jejunostomy should be made under local anaesthesia through a very small incision to the left of the umbilicus and with minimal disturbance of the bowel This is a life saving measure and decompression is usually followed by the normal passage of gas or a motion by the rectum and rapid diminution in the jejunostomy drainage Persistent copious jejunal drainage suggests organic obstruction and calls for exploration for its relief as soon as the patient is well enough Alternatively a tube of the Miller Abbott type may be introduced *via* the enterostomy and used to empty the more distal intestine

Finally it should be emphasised that in all the above examples as well as the observed losses the invisible loss of fluid and electrolytes into the gut may be very great indeed and the patient s condition may rapidly deteriorate into profound hypovolaemic shock Intravenous supportive therapy needs to be applied early and very thoroughly if lives are not to be lost

Internal herniation

In 1952 Stammers first drew attention to the danger of internal herniation following the Polya type of gastrectomy Such herniations usually occur after an antecolic anastomosis the efferent or afferent loop may pass mostly from right to left between the anastomosis in front and the transverse colon behind Subacute or acute obstruction results and the bowel may later become gangrenous Symptoms usually appear about the fifth or sixth post-operative day but sometimes the onset is delayed for months or even years Stammers (1954) states that the symptoms are not dramatic At first there is vague continuous upper abdominal pain and occasional vomiting only later does evidence of strangulation appear

Internal herniation can be avoided by keeping the afferent limb of a Polya gastrectomy fairly short (10 15 cm) Stammers (*loc cit*) suggests inserting some stitches to close the gap between the afferent loop and the colon and mesocolon If herniation has taken place the abdomen should be re-opened at once the gut reduced and steps taken to prevent a recurrence

Retrograde intussusception

This complication is rarely seen after Billroth I or Polya types of gastrectomy (one such was treated in the Liverpool Royal Infirmary this year 1959) but is less rarely encountered after gastro-enterostomy and after a Roux-en Y type of anastomosis (Irons and Lipin 1955 Bradford and Boggs 1958)

After gastro-enterostomy retrograde intussusception of one or both limbs of the jejunum into the stomach may occur usually many months later The condition may be acute with severe upper abdominal pain and vomiting but occasionally is subacute and recurrent When persistent the typical radiological appearance shown in Figure 33 makes the diagnosis clear Intussus

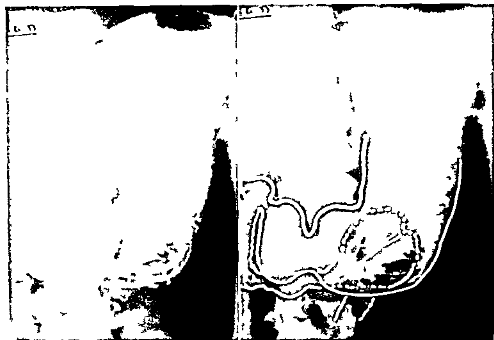


FIG 33
Jejuno gastric intussusception after a posterior gastro enterostomy

ception to this extent should be treated by operation and it is best to carry out a partial gastrectomy

Jejuno jejunal intussusception after the Roux-en Y type of anastomosis has been reported in a retrograde direction up the afferent loop (Davey 1954)

Food bolus obstruction

Following partial gastrectomy there is a slight but definite risk of obstruction of the small bowel by a bolus of imperfectly masticated or undigested food (Ward McQuaid 1950 Norberg 1955 Staven 1957 Butler 1959) Most of the patients so affected have had a Polya type of gastrec tomy but bolus obstruction can follow a Billroth I operation or simple gastro enterostomy Citrus fruits especially oranges are the commonest offending foodstuffs The bolus usually becomes impacted in the lower ileum and enterotomy is necessary for its removal A case seen recently developed acute

obstruction 72 hours after eating an orange and 13 years after his Polya gastrectomy

Jaundice

Any type of reaction after a blood transfusion is now rare in Britain Very occasionally when many pints of blood have been given before during or after an operation a slight icteric tinge may be detected during the post operative period This is a haemolytic type of jaundice and no bile is present in the urine As a result of transfusion also jaundice may develop about three months after the operation This is due to homologous serum hepatitis which is most clearly distinguished by the date of its onset The usual signs and tests of hepatitis are appropriate namely the presence of bile in the urine the frequently pale stools and the elevated serum transaminase level The condition is often alarming and may be fatal

Jaundice which develops a few days to a few weeks after gastrectomy however and which is obstructive in nature is a grave prognostic sign Obstructive jaundice after gastrectomy results from damage to or occlusion of the hepatic artery or common bile duct The hepatic artery may be damaged (especially if its origin from the coeliac axis is abnormal) when the left or the right gastric artery is being ligatured Alternatively the hepatic artery may be damaged during dissection round the antrum and duodenum Jaundice from this cause develops immediately and rapidly is associated with pyrexia and systemic collapse and usually with renal insufficiency as well as hepatic failure The blood urea rises urinary output is small peritonitis ensues as a result of infarction of the liver associated with anaerobic organism and death is not long delayed Supportive therapy and Neomycin (1 2 g six hourly) are indicated

A penetrating duodenal ulcer distorts the normal anatomical relationships in its vicinity The common bile duct may be drawn near to the ulcer and so become exposed to injury when the duodenum is being mobilised (Carpenter and Crandell 1958) The flow of bile can also be interfered with by excessive invagination of the duodenal stump during a Polya gastrectomy The resulting jaundice is typically obstructive in character A few mild cases presumably caused by oedema around the termination of the common duct clear up rapidly and require no special treatment Where the obstruction is complete and persistent operative repair or reconstruction of the extra hepatic biliary passages is necessary

Necrosis of the transverse colon

Rarely gangrene of the transverse colon results from damage to the middle colic artery This vessel lies considerably to the right of the midline

and the transverse mesocolon may be drawn in farther to the right as a result of penetrating ulcer of the duodenum or pyloric antrum In such an event the middle colic artery may lie very close to the ulcer The artery is however rarely damaged as it is always sought and preserved Damage is most likely to occur when a gastro-jejunostomy with stomal ulcer is being altered to a partial gastrectomy In any event the transverse colon and its vascular supply should always be examined at the end of any difficult operation of this kind Devascularised colon should be resected and continuity re-established by end to-end union

Should such a catastrophe pass undetected gangrene of the colon results and causes fatal peritonitis

Necrosis of the gastric remnant or great omentum

These are very rare or rarely recognised complications of gastrectomy To render the gastric remnant ischaemic requires a very extensive mobilisation of both curvatures such as may be required when resecting a juxta-oesophageal ulcer Only a few cases have been reported (Rutter 1953 Stuart and Jordan 1957) and the outcome has usually been fatal

Necrosis in or of the great omentum has been held responsible for mortality by Fritzsche (1942) who advises conservation of the gastro-epiploic arcade which is certainly to be recommended Kirschner and Garlock (1954) suggest that if the blood supply is impaired the omentum should be removed There is surprisingly little information in current literature about the fate of the omentum after mass ligature of the two ends of the gastro-epiploic arcade which is so frequently and probably inadvisably done Indeed areas of necrosis certainly occur from time to time even when the whole of the blood supply appears to have been conserved (Wells 1958 personal communication)

Volvulus of the stomach may follow excessive mobilisation while a redundant transverse colon may undergo torsion if the great omentum is removed (Stuart and Jordan 1957)

Oesophageal stricture and dysphagia

The development of an oesophageal stricture has been a recognised complication of abdominal operations for over twenty years but it is only recently that its aetiology has been elucidated The stricture develops a few weeks or months after an operation usually for peptic ulceration or rarely for cholelithiasis (McCredie and McDowell 1958) wherein gastric aspiration through a Ryle s tube has been carried out for some days after the operation It has now been shown (Bingham 1958 McKeown 1958) that in most if not all cases there is a hiatus hernia present causing reflux oesophagitis and

stricture formation The hernia may not have been recognised at the time of the original operation and peptic ulceration and cholelithiasis are the conditions most likely to be confused or associated with a hiatus hernia The *recumbent position of the* patient and the splinting and irritant effects of the red rubber nasogastric tube may aggravate but are not the cause of the oesophagitis

If the hiatus hernia is recognised before or at the time of operation for peptic ulcer a vagotomy with partial gastrectomy and Roux Y *anastomosis* (*Wells and Johnston* 1955) should be carried out Bingham (1958) suggests that any patient who requires gastric aspiration should if possible be nursed well propped up rather than lying flat A non irritant *plastic nasogastric* tube may be better than one of red rubber

After vagotomy a few patients experience slight difficulty in swallowing for three to four weeks (Bruce and Small 1959) *Reflux oesophagitis* without stricture formation can also cause dysphagia very rarely fibrosis around the lower oesophagus can interfere with swallowing

Post operative enterocolitis

Acute and often fatal inflammation of all or part of the small and large intestines developing within a few days of operation has been known for over sixty years but was seldom recognised in Britain until the paper of Bruce (1954) aroused interest in the subject

Aetiologically it is probable that several different conditions are being included under the non specific title of post operative enterocolitis but only one of these—staphylococcal diarrhoea—has been clearly differentiated from the others Clinically and pathologically pseudomembranous (necrotising) enterocolitis in which there is necrosis and sloughing of the intestinal mucosa forms a sufficiently striking picture to merit separate description although several different mechanisms may be responsible for the same pathological changes

STAPHYLOCOCCAL DIARRHOEA commences on the second or third day after an operation and is characterised by severe diarrhoea increased bowel sounds pyrexia and a painful tender and tympanitic abdomen (Webster 1958) Restlessness and *confusion* are common and there may be hypotension and tachycardia Pseudo-membrane formation is rare Most patients have had antibiotics before operation and on culturing their faeces an almost pure growth of antibiotic resistant coagulase positive staphylococcus aureus is *obtained* Gram negative organisms have practically or completely disappeared There can however be resistant staphylococci in the faeces without

serious symptoms their presence in the faeces has an ill understood relationship to their presence in the patient s nasopharynx (Penman and Pullan 1958) and a nasogastric tube may precipitate or hasten their entrance into the intestines (Webster 1958)

In treating staphylococcal diarrhoea the most urgent task is to replace the very large quantities of fluid and electrolytes especially sodium which are lost from the body One litre per hour may be requires for six to eight hours giving alternate litres of normal saline and five per cent dextrose (Webster 1958) with added potassium chloride up to say 5 g spread over each twenty four hours Whatever antibiotic has been given to the patient is stopped and erythromycin or novobiocin administered pending the results of sensitivity tests

PSEUDOMEMBRANOUS ENTEROCOLITIS —Acute pseudomembranous (necrotising) enterocolitis develops a few days after operation and its outstanding clinical feature is sudden circulatory failure rather than diarrhoea There is severe peripheral cyanosis and hypotension but in the series of cases described by Kay *et al* (1958) only one-quarter had marked diarrhoea Pyrexia and abdominal pain and distension are less common than in staphylococcal diarrhoea and the patient is mentally alert

The aetiology is obscure It has followed a wide variety of operative procedures and is occasionally a complication of purely medical diseases (Kleckner *et al* 1952) Recently however most examples have occurred after partial gastrectomy for peptic ulcer and enterocolitis is now one of the commonest causes of death after gastrectomy (Owen 1957) A fall in blood pressure has been blamed for the necrosis of the intestinal mucosa but parts of the intestine may escape and Hultborn (1956) has observed a case in which the necrosis of the mucosa did not extend beyond a colostomy Some but not all patients have received antibiotics or chemotherapy before the enterocolitis develops A variety of organisms including clostridium Welchii may be cultured from the faeces their exact types may in part depend on what organisms are prevalent in the wards Organisms however cannot always be isolated and Corridan and Shucksmith (1958) think that they may only have a secondary role There is however no doubt that pathogenic organisms may rapidly appear in the gastric stump and upper small bowel once gastrectomy has diminished the secretion of hydrochloric acid (Howie 1954 Hogman and Sahlin 1956) Perhaps as Pettet and her colleagues suggest (Pettet *et al* 1954) there may be many different causes giving rise to the same type of lesion in the mucosa of the small and large intestines

In the prevention of enterocolitis the routine administration of dilute hydrochloric acid to patients for a week after gastrectomy has lowered the incidence and mortality of post operative enterocolitis (Tanner and Owen 1958 personal communication) Another worthwhile prophylactic measure is to avoid indiscriminate antibiotic therapy and Illingworth and Kay (1957) have advised strongly against prophylactic antibiotic therapy in abdominal surgery Such therapy may permit the growth of resistant organisms capable of damaging the bowel An adequate blood pressure should be maintained during and after operation and abdominal distension prevented or dealt with effectively should it appear

Once pseudomembranous enterocolitis has developed treatment must be energetic, but is often unavailing (Kay *et al* 1958) Immediate efforts are directed towards maintaining the blood pressure and replacing lost fluid and electrolytes Large quantities of intravenous fluids are necessary and blood transfusion may be beneficial If the blood pressure remains low or falls in spite of these measures intravenous noradrenaline or hydrocortisone (p 128) is tried Oxygen is given whenever cyanosis is present If the patient has been receiving any antibiotic that antibiotic is at once discontinued and erythromycin neomycin or novobiocin given Sensitivity tests are carried out on the faecal organisms and any relevant indications are followed For fear that the sudden collapse of the circulation may be due to intestinal obstruction or strangulation—and clinically differentiation may be difficult or impossible —Kay and his colleagues (1958) advocate immediate laparotomy to ensure that no remediable lesion is missed

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CHAPTER VIII

PERFORATION

BY JOHN A SHEPHERD

ACUTE perforation of a peptic ulcer accounts for twelve per cent of all emergency operations in the abdomen During the last fifty years there has been a considerable increase in the incidence of peptic and especially of duodenal ulceration in the population The frequency of perforation increased to an even greater extent up until 1953 (Jamieson 1955) but since then perforations have become rather less common (Pulvertaft 1959 personal communication) In Liverpool eighty seven per cent of the patients are men the majority are between twenty five years and forty five years of age but no age group is exempt from the risk of perforation The disaster generally occurs towards the end of a working day rarely it may take place when the patient is in bed at night The effects of stress and other epidemiological factors are considered in Chapter I Speed in instituting definitive treatment is desirable too many patients still do not come under the care of the surgeon within six hours of perforating

Pathology

In the writer s series of cases the ratio of duodenal to gastric perforations is 7 1 At operation the oedema and inflammatory reaction frequently renders it difficult to determine on which side of the pylorus the perforation is situated In fact the vast majority of so-called juxta pyloric perforations are on the duodenal side (Yudin 1939) Ulcers situated on the antero-external aspect of the duodenum or on the anterior wall of the stomach are unsupported by adjacent structures and therefore perforate much more frequently than posterior wall ulcers which are more likely to penetrate and cause haemorrhage Duodenal perforations are only 3 4 mm in diameter but those in the stomach may be larger

Perforation may be the result of either a chronic ulcerative lesion or else of an acute ulcer which may later recur Kennedy (1951) reports that one in six gastric perforations occurs in carcinomatous ulcers In two to three per cent of cases two ulcers may be present and these may be of different chronicity One may bleed at the time when the other perforates very rarely they may perforate together

Bacterial contamination of the peritoneal cavity occurs at the time of perforation An infective inflammatory peritoneal reaction develops as a

result and the rate of development and severity of this process depends in part on the size and position of the perforation In general a gastric perforation gives rise to a grosser and more serious type of soiling than does a duodenal perforation

The sequence of events following perforation is as follows

There is a sudden escape of irritant gastric and duodenal contents which contaminate the adjacent peritoneum and viscera and which later may spread throughout the peritoneal cavity Irritation of the parietal peritoneum causes intense pain and protective rigidity in the overlying muscles There is an outpouring of fluid into the peritoneal cavity and also into the extraperitoneal tissues (Cope *et al* 1955) The bowel wall is quickly paralysed and soon flakes of fibrin are formed in the vicinity of the ulcer Bacteria are multiplying all the time but for the first few hours the peritonitis is chemical rather than bacterial

Generalised peritonitis may develop or if only a small quantity of gastric contents has escaped early localisation may occur The inflammatory process may be localised to the immediate vicinity of the ulcer sometimes gastric contents trickle down the right paracolic gutter and give rise to an abscess in the right iliac fossa

Late localisation takes place usually after operation through the accumulation of unabsorbed inflammatory exudate in some part of the abdominal cavity The common sites for such accumulations are in the sub phrenic spaces and in the pelvis Total empyema of the abdominal cavity is an uncommon sequel to perforation but is no longer incompatible with life

Recently Le Roux (1957) has drawn attention to the frequency with which scattered areas of collapse may be found before operation in the lungs of patients suffering from perforated peptic ulcer Many of these patients are heavy smokers with copious tenacious bronchial mucus The sudden inhibition of diaphragmatic and lower thoracic movement following perforation allows this mucus to plug the bronchi causing collapse and infection in the lungs

Clinical features

Usually there is a previous history of dyspepsia and the patient may know from earlier investigation that he has an ulcer It should be remembered however that a patient suffering from a perforation may be in no fit state to give an accurate history of previous indigestion Such information may only be obtained by further questioning during convalescence Rarely perforation may take place during a first attack of acute ulceration

An untreated patient passes through three clinical phases following perforation of an ulcer

The *initial phase* is heralded by sudden overwhelming abdominal pain at first epigastric but rapidly becoming more generalised It is usually plain to all that a major intra abdominal disaster has occurred The patient lies prostrated pale anxious and fearful of being moved Shoulder pain may be admitted on direct questioning

On examination pallor and sweating are mostly found respiration is rapid or grunting and thoracic in type the pulse may be rather fast but the blood pressure is normal—there is no true hypovolaemic shock present The abdominal wall is as rigid as a board and may, on respiration seem to rock like a see saw on a transverse line across the epigastrium The abdomen is often tympanitic from the escape of gas, liver dullness may be diminished or absent but this sign is less reliable than its radiological counterpart The patient s suffering is obviously so great that it is rarely justifiable to attempt to elicit shifting dullness Tenderness may be present on rectal examination

This phase usually lasts for three to four hours

The *deceptive phase* is rarely seen and is the more dangerous on that account for the most experienced clinician may fail to recognise it Peritoneal exudate separates the inflamed surfaces pain is lessened and the patient looks and feels rather better It should however be possible to detect some abdominal rigidity and distension shifting dullness and rectal tenderness are mostly present and an X ray should reveal free gas under the diaphragm

The deceptive phase lasts for a few hours but is frequently absent it may be closely simulated by the beneficial effects of intravenous fluids and morphia

The *phase of peritonitis* develops in from six to twelve hours after perforation and usually without any recognisable intermediate phase There is increasing general illness and toxicity The pulse rate rises and the temperature either rises rapidly or occasionally falls to an ominously low reading The pulse and temperature readings although helpful are not reliable guides to the patient s condition

With increasing paralytic ileus there is increasing abdominal distension and no flatus is passed Pressure on the diaphragm embarrasses respiration and adds to the circulatory and respiratory distress caused by the withdrawal of fluid from the circulation Later features of ileus such as effortless vomiting are seldom seen as untreated patients may not survive long enough for them to develop

A *self limiting perforation or leak* and a threatened perforation most often present as a severe exacerbation of pre-existing ulcer symptoms There is localised tenderness and guarding In such cases the occurrence of true perforation may remain in doubt or may be confirmed by X ray (see below) or by the subsequent development of an abscess This type of emergency is

unlikely to arise in a previously healthy individual because efficient localisation does not usually occur in the complete absence of previous inflammatory reaction

DeBakey (1940) has suggested that a correct diagnosis of perforation is made in almost ninety per cent of cases The writer in forty consecutive cases made thirty six correct pre-operative forecasts but wrongly diagnosed a perforated ulcer in six other patients during the same period These six patients however all had acute abdominal lesions requiring urgent operation The error of 10 per cent or more is an important factor in determining the choice between operative and non-operative treatment

Ancillary methods of diagnosis

A profile X ray of the anterior abdominal wall of the supine patient or a straight X ray of the diaphragm in the vertical position shows free gas if it is present in the peritoneal cavity Similarly small collections of gas may be shown in localised perforations Le Roux (1957) believes that a straight X ray of the chest of a semirecumbent patient is the best method of demonstrating subphrenic collections of gas the method also reveals any pulmonary lesions The absence of radiological signs of free intraperitoneal gas is strong presumptive evidence against a peptic ulcer perforation

It is not usually possible to distinguish between a perforated gastric and a perforated duodenal ulcer Moore (1955) has suggested the instillation of radio-opaque material by the indwelling gastric tube but pylorospasm may prevent the commonest site of leakage from the first part of the duodenum being shown The manipulations required for this additional diagnostic radiography are an added strain to a shocked patient and are rarely justified

Differential diagnosis

The most reliable guides to a correct diagnosis of perforated peptic ulcer are provided by a suggestive history of previous dyspepsia by the demonstration of gas under the diaphragm and by the persistence of abdominal pain rigidity and when present shifting dullness and rectal tenderness The conditions which may mimic perforated peptic ulcer can be grouped as follows

1 Those in which operation is in any case essential

2 Those in which operation is not essential but may not be harmful and may even be alternative and satisfactory treatment

3 Those in which operation is likely to be harmful

In the first group are a wide variety of perforated lesions of the gastro-intestinal tract and some gynaecological conditions Acute appendicitis by causing a sudden generalised peritonitis after perforation should not be

mistaken for perforated ulcer if the history is taken carefully and the maximal tenderness in the right iliac fossa recognised However a duodenal ulcer which leaks causing a collection of fluid in the right paracolic gutter, can give rise to a deceptive clinical picture almost indistinguishable from acute appendicitis At operation a gridiron incision may reveal an appendix with a hyperaemic serosa The true diagnosis should be suspected if the same degree of inflammatory reaction is present in adjacent bowel if on removing and splitting the appendix the mucosa is found to be healthy and if an odourless bile stained effusion is seen trickling down from the upper abdomen The local incision should then be closed and the perforation approached from above

Other conditions for which operation is urgently required include free perforations related to gastric carcinomas the rare perforations of duodenal or jejunal diverticula foreign body perforations of the gastro-intestinal tract and sudden unheralded perforation of uninflamed colonic diverticula Ectopic gestation with tubal rupture may cause acute signs and symptoms similar to those of perforated peptic ulcer but usually the effects of bleeding are apparent

In the second group the most important conditions to consider are acute pancreatitis and acute cholecystitis The perforation picture of some examples of acute pancreatitis may be very misleading The absence of free gas clinically and as shown by X ray is a helpful point While the signs and symptoms of perforated ulcer tend to be unremitting over a period of hours those of acute pancreatitis are fluctuating and there are curious exacerbations and remissions of shock vomiting pain and rigidity In the more fulminant types of pancreatitis true hypovolaemic shock and cyanosis are present from the start whereas in perforation they only appear late on in the phase of generalised peritonitis Severe back pain is usual in pancreatitis and rare in perforated ulcer The serum amylase tests may be deceptive as they are increased in both conditions although the increase is generally much greater in pancreatitis (Wapshaw 1949)

Acute cholecystitis may mimic perforated ulcer especially when the inflammation is of a fulminant type or when it occurs in association with torsion or perforation of the gall bladder Fortunately the confusing cases of cholecystitis are the very ones which require laparotomy *urgently* At the other end of the scale relatively mild acute cholecystitis is sometimes confused with doubtful duodenal perforation and entirely local peritonitis *In cholecystitis and appendicitis there is often a colicky quality about the pain in perforation the pain is continuous*

In the third group are the cardiac pulmonary pleural and diaphragmatic crises and inflammations which occasionally *present with abdominal signs as a temporarily dominant feature* In all these conditions the local signs in the abdomen are those of guarding which varies during respiration *True muscle*

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In the third group are the cardiac pulmonary pleural and diaphraematic crises and inflammations which occasionally present with abdominal signs as a temporarily dominant feature In all these conditions the local signs in the abdomen are those of guarding which varies during respiration True muscle

rigidity is lacking and abdominal tenderness is uncertain in location and relatively mild in degree By taking a careful history and by examining the chest as well as the abdomen such conditions as coronary infarct pulmonary collapse or consolidation should not be missed

In this group may be included the abdominal crises of tabes The pupils and knee jerks should be examined routinely However peptic ulcers are quite common in tabes (Schindler 1957) and it is probable that a perforation is more likely to be missed than to be diagnosed in error in a tabetic subject

Complications

The principal complications of and the chief causes of the mortality with perforated peptic ulcers are collapse and infection in the lungs and residual abscesses within the abdomen Scattered areas of collapse may be present in the lungs within a short time of perforation and before operation (Le Roux 1957) the tendency to pulmonary collapse and infection is increased by the abdominal distension and by peritonitis The commonest cause of a subphrenic abscess is a perforated peptic ulcer constant watch for residual abscesses must therefore be kept during the convalescent period

In recent years the incidence of these lethal complications has been greatly reduced by routine antibiotic and intravenous therapy and by modern techniques in anaesthesia with bronchial aspiration being carried out when required

TREATMENT

It is possible today to treat most cases of perforation within six to eight hours of onset and in general terms it can be stated that the greater the delay in starting effective treatment the higher will be the mortality rate The choice of treatment lies between non operative and operative methods Whatever the choice it should always be remembered that the patient is suffering one of the worst types of pain known to mankind and once the diganosis has been made he should be relieved from this pain as quickly as possible A slow intravenous injection of morphia gr $\frac{1}{4}$ or pethidine 100 mg can be given by the doctor and an intravenous saline drip should be set up These measures will frequently effect a considerable improvement in the patient s general condition In more advanced cases with peritonitis plasma or blood should be given An aspirating tube of adequate bore is passed routinely into the stomach and continuous or intermittent suction applied to it in order to prevent any further peritoneal contamination

There should be no delay in giving an antibiotic While a wide spectrum antibiotic like aureomycin or terramycin might seem the ideal form from the point of view of controlling a mixed peritoneal infection the danger

of serious side-effects from these drugs must be considered Of the existing preparations it is probably best to give an immediate pre-operative dose of 1 g of streptomycin followed by 0 5 g doses t i d for the first three days after which an antibiotic is only given if there is a clear indication for its use

Non operative treatment

Of the many advocates of non-operative treatment Taylor (1946 1951 and 1957) is a forceful representative Conservative or aspiration treatment is based on the assumption that clinical and radiographic diagnostic methods are accurate that leakage may be controlled or diminished by gastric suction that electrolyte and fluid loss can be corrected and that peritoneal infection is minimal in the early hours after perforation and can be controlled by antibiotics Taylor (1957) believes that this method facilitates natural healing processes whereas suturing of a perforation may make an acute ulcer become chronic and intractable

The details of a strict non operative regime are as follows

After the stomach has been emptied through a wide bore tube it is kept empty by intermittent or continuous suction through a small indwelling tube Twice daily X rays are taken to check the position of this tube and to ensure that the amount of free gas in the abdomen is decreasing Frequent clinical examinations are made by an experienced surgeon to ensure that the signs of peritonitis are diminishing Antibiotics and an initial dose of morphine are given as described above Watch is kept for any tendency to air swallowing a feature which if uncontrolled causes progressive leakage and a consequent failure of the method Gastric aspiration and intravenous fluids are continued for three to five days If under observation there is any reason to believe that the method is ineffective or the initial diagnosis wrong then recourse must be had to operation

The non operative regime should not be employed when there is any doubt about the diagnosis when the patient is known to be an air swallower or has had a heavy meal just before perforating

Non-operative treatment was advocated strongly about 1946 and onwards because of a belated recognition of the very high mortality (then fifteen to twenty per cent) in cases of perforation treated operatively In a collective review Taylor (1951) found forty-one deaths in a total of 454 cases treated by various surgeons Today operative mortality is very much lower and is still lessening This trend in the reduction of mortality is shown by Sangster (1948) who had twenty four fatalities out of a hundred cases in the decade up to 1939 and only four deaths in a hundred cases in the subsequent decade In a series of 277 cases reported by McCaughan and Bowers (1957) the overall mortality was 3 2 per cent and for simple suture the mortality was only 1 5 per cent

If the mortality and morbidity following non operative treatment were less than that after operative procedures there would be no argument about which method to employ Unfortunately the cases treated conservatively are often carefully selected and any comparison between the results of the two methods is difficult

Those who object to non-operative treatment stress the risk of error in diagnosis the inadequacy of aspiration by a tube in the stomach for a duodenal leak the difficulty of ensuring adequate radiographic control and frequent assessments by an experienced clinician and the high incidence of air swallowing when a gastric tube is *in situ* Without laparotomy the site of perforation is unknown and a perforated carcinoma of the stomach may not be recognised as such Although the risk of ordinary post-operative complications is eliminated there is still the possibility of a subphrenic abscess developing There remain however a few absolute indications for the adoption of non operative treatment carried out strictly according to Taylor s regime These are

1 Uncertainty of differentiation between perforated ulcer and a condition in which operation might prove dangerous *e g* coronary thrombosis
2 Lack of adequate operating facilities *e g* at sea
3 Refusal to submit to operation

Operative treatment

The choice of operative treatment lies between simple closure of the perforation and immediate gastrectomy Closure combined with gastro enterostomy once practised as a routine is now known to carry a high risk of recurrent ulceration and often early perforation Gastro-enterostomy is seldom justifiable on the grounds of pyloric stenosis having been caused by the duodenal sutures as with the subsidence of acute oedema in the region of the ulcer any initial delay in gastric emptying is soon rectified

SIMPLE CLOSURE —Simple closure is the standard procedure and one which is effected rapidly

The operation is done through a three inch or four inch midline supra umbilical incision Excess fluid and food fragments are removed by suction and the stomach gently drawn out to expose the pylorus near which the ulcer is almost always found Closure is effected by inserting a row of three to five catgut sutures which when tied appose two ridges of muscle and serosa in the transverse axis of the stomach or duodenum (Fig 34) If the ulcer edge is friable the stitches may have to be passed through all coats Reinforcement is hardly necessary but if used is best effected by anchoring over the sutured ulcer a live tag of adjacent omentum taking care not to strangle its vessels If the

ulcer is *gastric and gastrectomy* is not thought necessary a shaving of tissue should always be taken for microscopy Drainage is only required when there is excessive contamination of the peritoneal cavity with solid foodstuffs or when the delay in operating exceeds twelve hours and there is established purulent peritonitis In the doubtful case a drain in the right subhepatic space and a pelvic drain are brought out through a stab in the right iliac fossa so that adhesions of small intestine may as far as possible be avoided

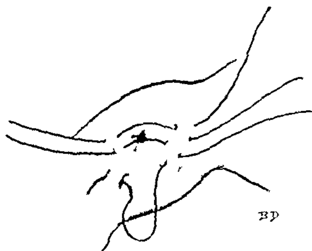


FIG 34
Simple closure of a perforated duodenal ulcer with three seromuscular stitches

There are many modifications of this simple procedure but anything which prolongs the operation or results in increasing displacement of the viscera is deleterious The essence of closure is gentleness and technical simplicity *If the perforation cannot be closed by suture omental plugging may suffice very rarely and in very ill patients it may be necessary to convert the perforation into a temporary gastrostomy or duodenostomy*

When no perforation is obvious in the duodenum or stomach adjacent to the pylorus the operator should then carefully examine the middle and upper parts of the anterior gastric wall especially up along the lesser curvature If the perforation still eludes detection the small and large bowel and the appendix should be inspected Only when perforated lesions in these viscera have been excluded should the lesser sac be opened and the posterior wall of the stomach carefully inspected free perforations into the lesser sac are very rare

Haemorrhage is a more frequent acute complication of stomal ulcer than free perforation (Davey 1959) Perforation of a stomal ulcer occurs more frequently after gastro-enterostomy than after partial gastrectomy Law (1940)

found the proportion 10 1 This is probably accounted for by the much greater frequency of stomal ulceration after a simple short circuit than after a gastric resection type of operation The signs and symptoms do not differ from those of perforated peptic ulcer at the common sites Simple suture is preferable to emergency gastrectomy in the gastro enterostomy cases as gastrectomy may be a prolonged and difficult procedure and should be done later as an elective operation If a stomal ulcer perforates as a sequel to partial gastrectomy simple closure is indicated and a vagotomy may be done later Toland and Thompson (1936) found a mortality of 17 6 per cent after simple closure of perforated stomal ulcer but in recent years the operative risk has been greatly diminished

Post-operatively gastric suction is continued in all cases until bowel sounds are heard again and until the aspirate is clear and small in amount During this period the patient is allowed to rinse out his mouth but nothing should be swallowed All fluids are given intravenously but if suction must be continued for more than thirty six hours it is reasonable to take the drip down each night to enable the patient to move and sleep comfortably Small quantities of water can be given hourly by mouth for a few hours before finally removing the aspirating tube All who are concerned with the treatment of the patient must encourage him to cough and to breathe deeply The services of a good physiotherapist are invaluable in helping to prevent complications and in expediting the patient s recovery

Prognosis after simple closure—Careful follow up investigations (Byrd and Carlson 1956 Matheson 1956 McCaughan and Bowers 1957) have revealed that only five to twenty five per cent of patients treated by simple suture are permanently cured Nearly fifty per cent continue to have symptoms and require treatment for chronic peptic ulceration About ten per cent—usually young subjects with duodenal ulcers—perforate a second time and from thirty to fifty per cent eventually require gastrectomy for intractable or obstructive symptoms

Emergency partial gastrectomy

Treatment of perforated peptic ulcer by partial gastrectomy has the great advantage of combining the emergency treatment of the perforation and the definitive treatment of the peptic ulcer in one operation Convalescence is often remarkably smooth and the patient is spared a prolonged and indefinite period of medical therapy and uncertainty about future surgery In skilled hands gastrectomy carries a lower mortality than simple suture In 1939 Yudin reported 937 resections with eighty four deaths Lowdon (1952) in a series of sixty five perforations had no deaths among fifty-one patients treated by gastrectomy four other patients not suitable for resection died an overall

mortality of six per cent When most perforations were treated by simple suture at St James Hospital Balham the overall mortality was seventeen per cent when more than half the perforations had gastrectomies performed the mortality was thirteen per cent and was only 3 4 per cent for those cases where resection was carried out (Desmond and Sergeant 1957) Gastric perforations however treated carry a higher mortality than duodenal perforations

Against the performance of partial gastrectomy as an emergency procedure are the facts that a major operation is being carried out in the presence of quite gross peritoneal sepsis and on an unprepared and at times shocked patient Furthermore a few patients have no further ulcer symptoms following simple closure of a perforation if all patients were treated by gastrectomy a few would lose their stomachs unnecessarily

Those who favour immediate gastrectomy stress that technically the operation is often simple In the presence of acute oedema the duodenal stump is freed much more readily than would be expected The emergency operation however should only be performed by a surgeon with considerable experience of gastric surgery and he should use the type of resection with which he is most familiar

It is generally agreed that gastrectomy should only be carried out in reasonably fit patients who have been perforated for not more than six to eight hours and who have already earned their gastrectomy by reason of prolonged severe ulcer symptoms or previous complications Perforations of acute ulcers or of stomal ulcers are best treated by simple suture (Desmond and Sergeant 1957) The mortality with simple closure of gastric perforations is so high (twenty five to thirty per cent) and the risk of underlying malignancy so considerable that many surgeons now treat all such gastric lesions by immediate gastrectomy Resection is undoubtedly the treatment of choice when perforation is associated with either haemorrhage or gross pyloric stenosis

The contra indications to immediate operation are fairly obvious Gastrectomy should be avoided when there is established purulent peritonitis and ileus or when there is some other serious disease Aged patients are not suitable subjects for prolonged emergency procedures and as they are much less liable to recurrent ulceration than the young and middle aged their perforations should be treated by a simpler method Emergency gastrectomy should not be attempted by those without considerable experience of abdominal surgery and should be avoided when adequate operating facilities are lacking

Summary

In summary it may be stated that early simple closure remains the treatment of choice for most perforated peptic ulcers although about fifty per cent

ents so treated will require further surgery Emergency partial gastrec in the hands of experienced surgeons offers an admirable alternative there are sound pre-existing reasons for resection and may be desirable a gastric ulcer has perforated Non-operative management has given results where there have been exceptional facilities and organisation for pe of treatment without special organisation no surgeon should adopt routine but it may be most valuable in unusual circumstances *e g* in at sea

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CHAPTER IX

BLEEDING PEPTIC ULCER

By John A Shepherd

MOST peptic ulcers probably bleed slightly at some time in twenty per cent bleeding may be sufficiently severe for the patients to vomit bright blood or to pass tarry or bloody stools A peptic ulcer usually in the duodenum is by far the commonest cause of serious gastro intestinal bleeding accounting for eighty per cent of such cases admitted to hospital (Tanner 1954) The patients are usually adult men the maximum incidence being in the forty to fifty age group (Chinn *et al* 1956) but it should be remembered that nowadays active duodenal ulcers are not uncommon in patients in their seventies

Pathology

A gastric or a stomal ulcer is even more liable to bleed than a duodenal ulcer but as duodenal ulceration is so much more prevalent four out of five bleeding ulcers are situated in the duodenum (Chinn *et al* 1956) Most of them are of chronic type but acute ulcers can give rise to serious haemorrhage Such acute ulcers may be multiple and non recurrent as in toxaemia uraemia and burns but frequently they are a variety of recurrent peptic ulceration The precipitating factor is mostly unknown Bleeding is commoner during cold weather (Harrison 1957) and in some cases it has followed trauma or operations such as prostatectomy (Goldfarb *et al* 1956 Rains *et al* 1957) Abrasive food particles may play a part

The source of the bleeding may be the hyperaemic mucosa around an ulcer or the granulation tissue in its base bleeding from these sources is generally slight Moderate or massive haemorrhage mostly comes from an eroded mural vessel or from an extragastric vessel such as the left gastric or gastro-duodenal artery which has been eroded by a chronic ulcer

The pathology of the blood vessels involved by penetrating ulcers is of interest (Osborn 1954) Such vessels may be seen at the edge of an ulcer or in its base with a widely open lumen and an almost erectile appearance This appearance is caused by oedema and fibrosis in the media and adventitia and is the result of inflammation not of arteriosclerosis True arteriosclerotic changes are unusual in the gastro-intestinal vessels even in the aged If a thickened radial artery is palpated it is not without significance however as it suggests that the patient s cerebral and renal vessels may be similarly affected and that he may be intolerant of arterial hypotension

Haemorrhage from a peptic ulcer is an index of the activity of the inflam matory process and at operation the signs of acute oedema hyperaemia and active regional adenitis are almost invariably observed In patients with multiple lesions one ulcer may be activated to cause haemorrhage and almost simultaneously another may perforate

SEVERITY OF HAEMORRHAGE —Bleeding from a peptic ulcer of immediate concern to the surgeon can be either

1 *Moderate*—bleeding sufficiently severe to justify immediate admission but there is no true hypovolaemic shock Persistent or recurrent bleeding of this grade may require surgical intervention within a day or two

or 2 *Massive*—history of acute severe bleeding with on admission evidence of hypovolaemic shock such as hypotension tachycardia pallor and sweating with some of the following signs

(*a*) Hb below 7 5 g per cent

(*b*) R B C volume less than sixty per cent of normal

(*c*) R B C count of 3×10 or under (Welch *et al* 1955 Mixter *et al* 1957)

Signs and Symptoms

Gastro-intestinal bleeding may be quite unheralded if the source is mucosal In the most dangerous type of bleeding from extragastric or extra duodenal penetration there is usually a history of chronic ulceration though the bleeding may occur during a period of symptomatic quiescence In a minority of cases serious bleeding is preceded by an aggravation of pain Although an inconstant warning sign back pain in a patient with a known duodenal ulcer should suggest the possibility of posterior penetration and the imminent risk of severe bleeding

Haemorrhage may present initially as haematemesis or melaena Vomit ing of pure blood or altered blood is copious and effortless and may be due to either gastric or duodenal bleeding Melaena the more treacherous presenta tion may be the only recognisable sign of gastric or duodenal bleeding and in severe cases of duodenal ulceration may rarely take the form of flooding of seemingly pure blood from the rectum

The degree of circulatory collapse depends on the rapidity of the blood loss as well as the total volume actually lost from the circulation With rapid and serious bleeding there develops a state of hypovolaemic shock with eleva tion of the pulse and respiratory rate and a fall in pulse volume and blood pressure The colour of the patient is at first only a rough guide to the extent of haemorrhage Subjectively dizziness weakness and malaise give some indication of the rapidity of the blood loss and may be the initial symptoms

before a melaena stool is passed Persistent headaches and dimness of vision indicate that the blood volume has been seriously depleted and that transfusion is a matter of urgency

The abdominal examination should be carried out with great gentleness Enlargement of the spleen and changes in the liver should be sought for Bowel sounds may be absent when the blood pressure is under 100 mm Hg when the blood pressure is normal the sounds are increased when clots are passing through the intestines and a sudden increase in the bowel sounds may be the first sign of a recurrence of bleeding (Milton and Clunie 1958) Retinoscopy may reveal evidence of pre-existing hypertension (Kennedy 1954)

The changes in the blood picture can be measured in various ways but only the most accurate and repeated plasma volume and haematocrit estimations afford any real guide to hour-to-hour variations (Gunz *et al* 1954) Haemoglobin and red cell estimations are of rather limited value as haemodilution may take several hours and the fall in haemoglobin and red cell counts after haemorrhage may not be demonstrable at first Of the observed biochemical changes the most important are the elevation of blood urea and the electrolyte disturbances following dehydration and—in some cases—sodium and chloride loss from vomiting A blood ammonia level above 100 mg per 100 ml suggests that the bleeding is really due to portal hypertension However after prolonged haemorrhage with dehydration and hypotension there may be renal and hepatic failure

Differential diagnosis

In a general survey Aird (1957) has derived the figure of eighty five per cent as representing the incidence of peptic ulceration as a cause of moderate and massive gastro intestinal haemorrhage In a personal series of 1 490 cases with severe bleeding Tanner (1954) found peptic ulcer to be the cause in eighty per cent and Avery Jones (1957) experience supports this figure In five per cent of cases the cause of the bleeding was uncertain while three conditions comprised the majority of the remaining fifteen per cent—acute gastritis carcinoma of the stomach and portal hypertension In addition there are of course many rarer causes of gastro-intestinal bleeding such as hiatus hernia naevi simple tumours and diverticula of the stomach duodenum and small intestine mucosal tears at the cardio-oesophageal junction (Mallory and Weiss 1929) lesions of the head of the pancreas aneurysms eroding into the gastro-intestinal tract and the haemorrhagic diseases Of these rare conditions only the blood diseases may be regarded as of important diagnostic significance In such cases laparotomy may well prove disastrous but in the other rare conditions operation is usually the

best treatment The presence of purpuric spots or of enlarged lymph glands or spleen should suggest the correct diagnosis A purpuric eruption may first be detected on the arm just after taking a blood pressure reading

Acute gastritis may give rise to slight or moderate bleeding and Tanner (1954) actually found it to be a slightly more frequent cause of haemorrhage than portal hypertension In some cases the patient may be suffering from septicaemia uraemia or a specific fever or there may be a history of ingesting irritants such as excessive amounts of alcohol or aspirin If none of these factors is at work then it may be very difficult to separate so-called acute gastritis from peptic ulceration of the acute type and salicylate preparations may in fact often precipitate bleeding from a peptic ulcer (Alvarez and Summerskill 1958)

Bleeding from portal hypertension offers a difficult problem both in diagnosis and management About one-quarter of truly massive haemorrhages from the upper gastro-intestinal tract are due to portal hypertension (Welch 1958) The source of bleeding is a ruptured oesophageal or gastric varix or ulceration of the hyperaemic gastric mucosa peptic ulcer and portal hyper tension frequently co-exist (Lipp and Lipsitz 1952) The portal obstruction may be due to hepatic cirrhosis or to some less common lesion Examination may reveal evidence of liver disease such as spider naevi on the face erythema of the eminences of the palm and changes in the nails The spleen is usually though not necessarily palpable While some cases of bleeding varices are amenable to surgical attack operation is better avoided in the acute phase If in doubt it is better to explore the abdomen to find evidence of portal hypertension than to miss an ulcer which is amenable to surgical treatment

Of the three common diseases which have to be differentiated from bleeding peptic ulcer carcinoma of the stomach is the one least likely to cause serious bleeding Although at any period of its development carcinoma of the stomach may cause recognisable bleeding massive haemorrhage for which operative intervention may seem urgently indicated usually occurs with advanced ulcerative or penetrating lesions At operation the large majority of such cases prove to be inoperable or at the best amenable only to some palliative procedure

In summary it must be stressed that in doubtful cases the cause of severe acute gastro-intestinal bleeding must be considered to be peptic ulceration until proved otherwise by exploration—this is a permissible interpretation of well-established statistical evidence

Prognosis

The decision whether or not to operate for bleeding peptic ulcer is based on a knowledge of the results to be expected from different lines of treatment

Unfortunately it is often impossible to compare such results The terms used to describe the severity of the haemorrhage vary from one hospital to another as does the type of patient admitted and individual physicians have their own particular modifications of medical treatment Good results were obtained twenty to thirty years ago in the treatment of bleeding ulcer but they should not be used as a basis for comparison with results being reported today Too many factors have altered in the intervening quarter of a century In the earlier period blood transfusion was less readily available dehydration and malnutrition were not so well understood and there were no antibiotics to combat the frequently fatal chest infections In spite of the development of these supportive measures there has not however been any dramatic fall in the mortality of bleeding ulcer in recent years (Avery Jones 1957) This rather disconcerting fact is probably accounted for by the ageing of the population and the increased incidence of active duodenal ulceration in elderly patients

At the present time the mortality with non-operative treatment in patients requiring blood transfusion is from ten to fifteen per cent The best results so far obtained have emanated from gastro-intestinal units where surgeon and physician work in the closest co-operation With early surgery in selected cases Ogilvie *et al* (1952) achieved a mortality as low as five per cent but a more representative figure would be the 11 7 per cent of Mixter *et al* (1957) It might seem too much to expect a mortality for emergency gastrectomy to be as low as that obtained for elective operations but such figures have in fact been produced where a high proportion of the patients have been seen early by the surgeon (Amendola 1952) Unfortunately many surgeons are denied this advantage and have to operate on a large proportion of nearly moribund and often elderly patients

The age of the patients has a considerable influence on the mortality to be expected from bleeding peptic ulcers Chinn *et al* (1956) studied the results of treating initial major haemorrhages from duodenal ulcers With patients under fifty years of age the mortality rate was six per cent whereas for patients aged fifty years and over the mortality was more than doubled —fourteen per cent In large series of patients treated rather differently Stewart *et al* (1956) and Avery Jones (1957) obtained similar results The difference was not quite so marked with second and subsequent haemorrhages Many older patients have serious associated diseases especially of the cardio-vascular system No prognostic significance can be attached to the length of time the patient has had ulcer symptoms prior to the onset of bleeding In twenty per cent of fatal haemorrhages there is either no history or else one of less than two weeks duration (Shapiro and Schiff 1952)

Gastric ulcers carry a worse prognosis than duodenal ulcers with gastric ulcers twenty two per cent of patients under fifty years and forty-eight per cent over fifty years die from their first haemorrhage (Chinn *et al* 1956) In elderly patients however there may be little difference between gastric and duodenal ulcer mortality rates (Avery Jones 1957) Naturally the larger the amount of blood lost the greater the mortality With massive haemorrhages (as defined on p 157) the overall mortality is about seventeen per cent while with moderate haemorrhages only three per cent of the patients die (Welch *et al* 1955) Haematemesis carries the same risks as melaena alone (Shapiro and Schiff 1952)

Although during the first few days after the patient s admission the physician and surgeon are mainly concerned with preserving life their decision for or against surgical intervention may be influenced by the risk of later haemorrhages Of patients treated medically who survive an initial haemorrhage from a duodenal ulcer about one in three bleed again maybe years later The risk is doubled after a second haemorrhage (Chinn *et al* 1956) but the chance of dying from later haemorrhages is less than with the earlier ones

MANAGEMENT DURING PERIOD OF OBSERVATION

A good history must be obtained from the patient or a relative as soon as possible Particular enquiry must be made about symptoms suggestive of chronic ulceration or of liver disease Direct questions should be asked about possible causes of gastritis and any bleeding tendencies and an attempt should be made to determine whether or not the patient was anaemic or hypertensive before the haemorrhage

The details of management during the critical day or two after initial bleeding are as follows

On admission blood is taken for typing and for determination of the haematocrit and of haemoglobin and blood urea levels The patient is then cross matched and steps are taken to ensure the ready availability of at least six pints of blood Grouping must be done before any emergency infusion of dextran or other plasma expander is given as these may confuse cross matching The patient is given morphine as this allays restlessness and apprehension A duodenal tube is passed and samples aspirated at hourly intervals without attempting to empty the stomach The pulse rate is charted half hourly and frequent observation of the blood pressure is most important Repeated clinical assessments by the physician and surgeon are of the greatest value

Prolonged periods of hypotension and anoxia must be avoided Unfortunately patients vary considerably in their response to haemorrhage and it is not possible to give indications for transfusion that are applicable in all

cases Catastrophic haemorrhage obviously requires urgent transfusion and most clinicians give blood if the pulse rate is rising steadily, and the patient becoming more distressed The very variable reactions of the cardiovascular system to blood loss and the length of time taken before haemodilution becomes stabilised render blood pressure and blood concentration readings rather unreliable guides as to when transfusion is required Bearing in mind the possible fallacies in such readings however transfusion is usually advised if the haemoglobin falls to fifty per cent or less if the systolic blood pressure is below 90 mm Hg or if the blood urea rises to 90 mg per cent or over

When transfusion is considered necessary the surgeon if not already consulted should certainly be asked to see the patient At least two pints of blood should be given over a period of two to three hours and on the response to this initial transfusion the need for continued blood replacement is determined In few cases does the degree of circulatory collapse demand that the drip should be accelerated Rarely however the patient fails to respond to a two to three pint transfusion given over a period of several hours and such failure indicates the need for accelerating the transfusion and preparing for operation as a matter of extreme urgency

In the majority of cases the blood can be followed by a slow glucose saline drip to maintain the patency of the apparatus Haematocrit and haemoglobin estimations should be performed several times per day and rectal examinations may give some information about continuing melaena It is wise to give penicillin routinely in the constant expectation of subsequent complications in the respiratory tract Vitamins should also be given as some of the patients may have vitamin deficiencies (Tanner 1957)

Initially the patient may lie flat without a pillow but as soon as there is a response to transfusion he should be progressively raised to three or four pillows This together with other changes of posture lessens the risk of chest complications especially in the aged In all patients the dizziness or faintness experienced on raising of the head is a valuable sign of recurrent bleeding Sips of water may be allowed from the start The conservative regime is continued in the absence of further evidence of bleeding milk may be given to drink and after three to five days the patient is transferred to a Meulengracht or Witts regime of feeding with citrated milk and the accepted protein reinforcement

Under the conservative regime outlined above bleeding will cease in about three-quarters of the patients affected These patients can then be carefully investigated at leisure the fact that they have had a serious haemorrhage will be a strong argument in favour of performing an elective gastrectomy at a later date Persistence or recurrence of bleeding while under

treatment in hospital indicates that the bleeding is unlikely to cease until the responsible lesion is dealt with surgically

Early radiological examination is being advised increasingly frequently in cases of bleeding peptic ulcer (Welch *et al* 1955 Stewart *et al* 1956 Osborne and Dunphy 1957 Rigler 1959) In patients not previously investigated a barium meal may give valuable information about the source of the haemor rhage but the examination cannot be carried out until active vomiting has stopped and hypovolaemic shock has been corrected Gastroscopy can be useful in localising bleeding lesions in the stomach (Palmer 1952 Welch *et al* 1955) Even a skilled gastroscopist may however have difficulty in getting a clear view in a stomach full of blood and clot and the necessary manipulations by the relatively unskilled may be more dangerous than helpful

Indications for early operation

Most of the indications for operation within a day or two of admission to hospital have already been mentioned when considering the prognosis and management of bleeding peptic ulcer In many cases the decision for or against operation is based on several factors Only a few special circum stances such as catastrophic haemorrhage constitute a single clear-cut indication for operation The decision regarding the advisability of surgery is preferably made by the physician and surgeon working together and it is better made early—within twenty four to forty-eight hours—rather than late The main factors to be considered when making the decision are summarised below

Age of patient —Haemorrhage from peptic ulcer is associated with a high mortality in patients over fifty years of age Such patients frequently have other diseases they withstand periods of hypotension very badly and their response to transfusion may be poor Operation should therefore be advised earlier and more often than in younger patients A young patient with hypertension may however require urgent surgical treatment

Expected pathology —If it is known or suspected that a patient has a gastric ulcer operation is usually advisable A bleeding gastric ulcer is more likely to kill the patient than is a corresponding duodenal lesion A gastric ulcer often occurs in a patient whose general condition is poor and the ulcer is liable to erode into a major extragastric artery causing massive haemorrhage

Haemorrhage is a common presentation for a stomal ulcer especially if it develops following a gastroenterostomy (Davey 1959) When a stomal ulcer appears after a gastrectomy melaena is much more common than haemate mesis As far as bleeding is concerned a stomal ulcer behaves in a similar manner to a duodenal one Catastrophic haemorrhage may rarely take place

from an eroded middle colic artery but in general the indications for urgent surgery are the same as in the case of a duodenal ulcer Emergency gastrectomy may be technically difficult when there is an old retrocolic anastomosis

The chronicity of the ulcer must also be considered An ulcer which has been troublesome for many years is rather less likely to respond to conservative treatment than an acute lesion and early operation is more often indicated The same is true when a definite ulcer crater can be demonstrated radiologically (Gardner and Baronofsky 1959) Urgent gastrectomy may sometimes be necessary to save the life of a patient with an acute ulcer otherwise operation should be avoided Having had few or no symptoms before their haemorrhage patients with acute ulcers are frequently dissatisfied with the results of surgery on account of minor post gastrectomy symptoms (Pulvertaft 1958)

Amount of blood loss—The patient's or the relatives estimate of the amount of blood lost is notoriously unreliable A graphic description of vomiting pints of red blood cannot be ignored but the history is often vague and unhelpful Careful clinical assessment is much more important The loss of large quantities of blood suggests that a sizable vessel is bleeding and the larger the vessel the more likely is surgery to be necessary But small superficial ulcers can bleed profusely Catastrophic haemorrhage such as may follow erosion of the splenic artery or failure of blood replacement to keep pace with blood loss are the only indications for performing emergency gastrectomy before the blood deficit has been largely corrected Sometimes difficulties in obtaining adequate amounts of blood of the correct group for transfusion may make early operation advisable

Persistence of bleeding—When bleeding does not stop within twelve to twenty four hours under medical treatment operation is usually advisable Not infrequently it is difficult to decide when the bleeding has stopped It may be revealed by failure to aspirate blood from the stomach (Chandler and Watkinson 1953) Conversely persistence of bleeding may be shown by a gradually falling blood pressure and rising pulse and respiratory rates These changes are particularly serious when they occur despite transfusion If moderate bleeding continues [illegible] interruption [illegible] four hours operation should be performed [illegible] waste of

Recurrence of bleeding [illegible] intestin[illegible] of the type most likely to require opera[illegible] often [illegible] es of brisk episodes Each haemorrhage [illegible] in blood [illegible] with this fall bleeding may stop [illegible] blood [illegible] until the pressure [illegible] In [illegible] pre[illegible] ent [illegible] is a

of recurrence are not immediate and restlessness and circulatory collapse imply that a further haemorrhage is already well advanced A single recurrence of haemorrhage while in hospital strongly suggests that the ulcer is not going to be controlled by medical measures alone and that surgery is required

Coincidental complications—Of these the most important is perforation and the addition of the signs of perforation to those of acute bleeding calls for emergency gastrectomy (Moore and Fuller 1959) Bleeding in a patient known to have pyloric stenosis is also best dealt with by operation Pain by itself is not an indication for surgery in bleeding peptic ulcer The pain may form a rough guide to the activity and chronicity of the ulcer but it can be closely simulated by the pain of myocardial ischaemia resulting from the haemorrhage (Amendola 1952)

The localisation of the bleeding source at operation

While a large majority of peptic ulcers are easily found at operation a minority are not in the common sites A chronic ulcer which is obvious to palpation and inspection may not be the source of acute bleeding It is imperative that the surgeon should evolve for himself a plan of action the adoption of which will save valuable time and ensure that the source of bleeding is not missed

A midline epigastric incision extending from the xiphisternum to the umbilicus and continuing down for about an inch to the right of the umbilicus allows an adequate exposure As the incision is being made any defects in blood coagulation or the presence of dilated veins are noted On opening the peritoneum free fluid may escape or carcinomatosis be obvious while a cirrhotic liver margin may be visible in the upper part of the incision In most cases none of these abnormalities is found and so the duodenum is inspected and palpated first as it is the most common site of a bleeding ulcer The ulcer is usually situated posteriorly and its crater may be felt by invaginating the anterior wall of the first part of the duodenum In cases of chronic ulceration external scarring and fibrosis of a posterior ulcer spreads widely and distorts the pylorus If there is no evidence of duodenal ulceration the anterior wall of the stomach is inspected and palpated—particularly along the lesser curve The large penetrating chronic gastric ulcer is unmistakable but large shallow acute mucosal gastric ulcers may be difficult to feel If no ulcer is found the lesser sac is opened for palpation and inspection of the posterior wall of the stomach and bimanual palpation of the first part of the duodenum Failing the discovery of an obvious ulcer at this stage the possibility of other diagnoses must be considered The liver is inspected for cirrhotic changes the spleen palpated for abnormal enlargement and the rest of the duodenum examined

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Recurrence of bleeding—Serious gastro-intestinal bleeding of the type most likely to require operative intervention often occurs in a series of brisk episodes *Each haemorrhage causes a fall in blood pressure* and with this fall bleeding may stop spontaneously and a temporary clot may delay further blood loss until the pressure mounts once more In a few cases pain may precede a recurrent haemorrhage Unless there is a haematemesis the signs

of recurrence are not immediate and restlessness and circulatory collapse imply that a further haemorrhage is already well advanced A single recurrence of haemorrhage while in hospital strongly suggests that the ulcer is not going to be controlled by medical measures alone and that surgery is required

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Finally the oesophageal hiatus and the small intestine are palpated thoroughly It is important to remember that tumours and diverticula of the small intestine can cause haematemesis (Orr and Russell 1951) In few cases does a complete search fail to reveal the source of bleeding but on occasion a superficial mucosal or high lesser curve ulcer or an acute erosive gastritis defy discovery by external examination In such cases some surgeons make a long gastro-duodenotomy to inspect the interior of the stomach and duodenum (Welch et al 1955 Osborne and Dunphy 1957 Tanner 1958) The author however prefers to divide the pylorus after ligation of the greater curvature vessels to the distal half of the stomach and ligation of the right gastric vessels The open duodenum is then inspected and it is noted whether or not fresh blood or clot wells up from the cut end A finger is inserted into the duodenum and the first and second parts palpated directly In the absence of a duodenal source of bleeding it is reasonable to assume that the origin of bleeding is gastric and to proceed to a partial gastrectomy The stomach is divided at the usual level after ligation of the left gastric artery and the highest gastro-splenic branches The resected specimen is immediately opened and whether or not an obvious bleeding source is seen the upper gastric remnant is then palpated from within and visualised as completely as possible By adopting this method the risk of missing a high superficial gastric ulcer is eliminated The very few cases in which no bleeding point is identified at operation or by subsequent macroscopic and microscopic inspection of the excised tissue are almost always managed successfully by this blind gastrectomy

The choice of operative procedure

The primary aim is to stop bleeding and to save life by so doing The secondary aim is to cure the patient of the disease which has led to bleeding namely chronic or recurrent peptic ulceration The ideal operative procedure is a partial gastrectomy which removes the source of bleeding and leaves not only healthy gastric and duodenal tissue sutured safely and anastomosed appropriately but offers reasonably certain control of peptic ulceration

A mobile and easily resectable ulcer of the stomach or duodenum presents no great problem and the surgeon will adopt the form of gastrectomy to which he is accustomed The resection of a chronic gastric ulcer even when large and fixed is not likely to prove difficult There are however two special cases that merit special consideration

The first is the chronic gastric ulcer with torrential haemorrhage from a vessel outside the stomach wall The anterior wall of the stomach should be opened and the ulcer margins underrun so as to tent the ulcer site into the wound as much as possible The bleeding vessels are then identified and

underrun with sutures Time should then be taken to correct the general condition and to make preparations for performing gastrectomy Only in the most desperate circumstance should the ulcer be left exposed to the gastric content

The other problem is that of the large chronic duodenal ulcer In severe haemorrhage this is usually contained in a forbidding inflamed and oedematous mass Torrential haemorrhage may be controlled for the moment in the way just described by suturing across the opened duodenum It is however not wise to leave the ulcer exposed to the duodenal content even with closure of the stump (as in a Polya gastrectomy) and satisfactory closure may of course be exceedingly difficult to achieve Probably the best solution is to adopt the modified Billroth I procedure (described on p 115) making full use of the lesser curve end of the divided stomach for generous tamponade or even exclusion of the ulcer cavity The vagotomy may be postponed for a couple of weeks and then performed across the chest—a relatively minor procedure

The other method is that of Yudin (described by Gordon Taylor 1946) in which the lateral wall of the duodenum proximal to the ulcer is curled up like a snail s shell and tucked firmly into the ulcer cavity as a tampon The operation is then completed as a Polya procedure There is little or no information available about the efficacy of Yudin s manoeuvre

If the situation is desperate and the divided duodenum cannot safely be closed it is best to suture a catheter or small tube firmly into its lumen and to bring this out to form a temporary duodenal fistula Ligation of feeding vessels as a method of dealing with bleeding points is unsatisfactory and it is seldom that even temporary control of bleeding can be obtained in this way

In conclusion it may be said that when surgery is necessary the majority of cases of bleeding peptic ulcer should be managed by partial gastrectomy and that the ulcer crater should not be left in contact with duodenal or gastric secretions unless this alternative cannot possibly be escaped In the vast majority of patients partial gastrectomy will stop bleeding which is coming from a gastric or duodenal lesion but in rare instances serious bleeding may persist despite a partial resection and if no haematological abnormality is demonstrable these unfortunate patients may require an almost total gastrectomy leaving only a small fringe of gastric tissue around the cardia (Tanner 1958)

Donaldson *et al* (1958) have recently carried out a five year follow up study on patients treated for bleeding duodenal ulcers Of those not operated on forty per cent bled again within five years but over twenty per cent of those who did have emergency operations also bled subsequently and 12 5

per cent developed stomal ulcers Gardner and Baronofsky (1959) have produced similar figures half of their medically treated patients and 18 5 per cent of those treated surgically (mostly by Polya gastrectomy) had later haemorrhages It is important that any emergency gastrectomy should be as radical as the equivalent elective procedure if the risk of recurrent ulceration and bleeding is to be avoided

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CHAPTER X

PYLORIC STENOSIS

By Ian W MacPhee and James Kyle

In peptic ulceration narrowing of the pylorus or adjacent duodenum sufficiently severe to cause gastric retention may be due either to spasm and oedema or to organic contracture and scarring Both conditions are called pyloric stenosis both are due to an active peptic ulcer and they frequently co-exist in varying proportions From two to four per cent of patients with peptic ulcers finally develop stenosis (Balint and Spence 1959) Organic stenosis causes considerable dilatation of the stomach and stasis ulcers may develop (Johnson 1955) Although there is usually some super ficial gastritis present the mucosa is generally capable of secreting large quantities of hydrochloric acid Pylorospasm is usually caused by a duodenal ulcer or the closely related type of ulcer which occurs in the pyloric canal (Johnson 1957) It can however be caused by a gastric ulcer situated some distance from the pylorus (Eusterman and Balfour 1936) The effects of the spasm may be magnified by inflammatory changes and oedema in the pyloric mucosa Medical measures including gastric lavage may be surprisingly successful in enabling the oedema to subside and the muscle to relax with complete relief of an apparently hopeless obstruction

Clinical features

Most patients developing pyloric stenosis give a history of peptic ulcera tion of many years duration Vomiting has recently replaced pain as the main complaint The vomitus is sour smelling and contains undigested food but it is unusual for the patient to vomit after every meal With pylorospasm an ulcer type of pain may persist with organic obstruction pain is also common The patient experiences a feeling of gross distension and acute discomfort immediately after meals and may endeavour to relieve himself of this unpleasant sensation by deliberately inducing vomiting There is a steady and often marked loss of weight and appetite in two-thirds of the cases

The clinical state of a patient with pyloric stenosis depends on the severity and duration of the vomiting and on the relative proportions of water and the various electrolytes being lost in the vomit The losses resulting from

vomiting are complex as are also their consequences An attempt must however be made to analyse these consequences in terms of specific deficiencies (discussed in detail on p 172)

Sometimes if vomiting has only just started the patient may not appear ill Often however he is thirsty with a dry dirty tongue His cheeks and eyes are sunken and his skin slack and inelastic There may be cyanosis and rarely the patient is drowsy and irritable resenting examination Constipation is usual but diarrhoea occurs in twenty per cent (Balint and Spence 1959) only a small quantity of concentrated acid urine is passed In many patients there are signs of malnutrition and anaemia and oedema of the abdominal wall is sometimes present (Parsons and Watkinson 1954) The outline of the dilated stomach can frequently be seen and palpated The stomach can sometimes be rendered more prominent by giving the patient one or more tumblers of soda water to drink (Lees 1956) but usually it is so capacious that the swallowing of a small quantity of aerated fluid has little effect upon its physical characteristics Visible peristalsis is often present and a succussion splash can be elicited many hours after food in over half the cases

Diagnosis

Pyloric stenosis can be diagnosed on the history and clinical findings alone Barium meal examinations though usually done are seldom necessary *and the barium may have to be washed out in order to prevent its forming* mortar like masses in the stomach The radiological differentiation between *a simple and a malignant lesion causing pyloric stenosis may* be difficult or impossible In case of doubt the history is a more reliable guide and the finding of a *really large and hypertrophied stomach* with strong peristalsis visible is much in favour of a benign lesion

General management of pyloric stenosis

Patients with pyloric stenosis usually have an active duodenal or pyloric canal ulcer Although some recover temporarily from an obstructive episode without operation they later suffer from increasingly severe ulcer symptoms It is better therefore to spend one to two weeks preparing these patients for definitive surgery once they have recovered from their acute attack *The* main danger to life during an acute episode is the gross disturbance *of fluid* and electrolyte balance which may occur consequently the major *portion* of this chapter is devoted to a detailed study of these disturbances and to their correction

The general measures employed in treatment are designed to improve the local conditions in the stomach to correct deficiencies and to treat the ulcer

GASTRIC ASPIRATION —A tube of adequate bore is passed into the stomach and aspirated every half hour The standard size of rubber Ryle s tube is not really large enough to empty the stomach properly in a patient with pyloric stenosis A plastic or synthetic naso-gastric tube with an internal diameter of 4 mm (No 18 Charriere gauge) is better so long as it does not irritate the nasopharynx The stomach is irrigated twice daily with Ringer s solution or ½ N saline Bicarbonate of soda (approximately one to two per cent solution) is occasionally employed to rid the stomach of viscid mucus but many surgeons believe that plain water or bicarbonate solutions should not be used because of the possibility of increasing the electrolyte disturbance by dialysis through the gastric mucosa

Once the acute episode is over the aspiration tube is withdrawn but should be passed again each evening and the stomach emptied and washed out This enables the stomach wall to regain its tone

FLUIDS AND DIET —During the first twenty four to forty-eight hours after admission many patients receive intravenous fluids and can take nothing by mouth An accurate fluid balance chart must be kept Once oral feeding can be resumed a generous liquid diet should be given The patients are frequently undernourished and vitamins and iron should be added to the diet A blood transfusion is often valuable since a generally undernourished and dehydrated state such as is found in pyloric stenosis is constantly associated with true hypovolaemic anaemia On account of haemoconcentration blood analysis may however reveal no abnormality

ULCER THERAPY —The causative ulcer should initially be treated with sedatives and antispasmodics Belladonna is just as effective as many of the newer preparations but probably has more undesirable side effects An intragastric milk drip may sometimes be necessary but soluble alkalis must be avoided on account of the danger of alkalosis

The very active intractable type of peptic ulcer which causes pyloric stenosis should eventually be treated by radical surgery Gastro-enterostomy alone is quite inadequate and is associated with a very high incidence of recurrent ulceration In most cases a week or two can properly be spent preparing the patient for operation but sometimes with persistent complete obstruction operation may be necessary within forty-eight to seventy two hours as soon as the gross fluid and electrolyte derangements have been corrected

FLUID AND ELECTROLYTE IMBALANCE

Some patients show no evidence of dehydration and do not require any special replacement therapy. Other patients with only *mild symptoms* due to pylorospasm may respond rapidly to gastric lavage and antispasmodic drugs. They can soon take a generous fluid diet thereby correcting for themselves any minor disturbances of fluid and electrolyte balance from which they may have suffered. Only a few patients are moderately or severely *dehydrated and show evidence of electrolyte losses*. It is this group of patients which is considered below.

WATER LOSS.—Large quantities of *water* may be lost by vomiting. A normal person secretes about one and a half litres of saliva and two and a half litres of gastric juice per day (Gamble, 1947) and a patient with a duodenal ulcer may secrete an even larger volume of juice. This rate of secretion, however, is not maintained indefinitely in pyloric stenosis because of the effects of dehydration. But initially a patient with complete pyloric obstruction may lose as much as four litres of fluid in a day by vomiting. Furthermore no fluid reaches the intestines to replace the so-called metabolic loss through the kidneys and skin (normally two and a half litres per day). To replace the lost fluid and maintain the blood volume, water is withdrawn from the interstitial tissues and more particularly from the cells themselves.

WATER DEFICIENCY.—*Thirst is the cardinal symptom of intracellular dehydration.* The urine is scanty and in severe cases the patient may be confused and drowsy. Haemoconcentration may be present but only in extreme instances is peripheral circulatory failure due to pure water loss.

CHLORIDE LOSS.—Gastric juice contains *chloride* ions in a concentration fifty per cent greater than that in the plasma. Normally and in gastric hypersecretion most of the chloride is in the form of HCl, but in progressive pyloric obstruction the concentration of free acid in the juice gradually falls. Analysis then shows that the Cl ions are combined with Na and K rather than with H. Thus, *even though the gastric acidity has decreased*, chloride ions are still being lost in the vomit. In the blood some of the chloride ions are replaced by bicarbonate ions and alkalosis may result.

CHLORIDE DEFICIENCY.—The immediate effect of loss of chloride in excess of base is alkalaemia, *i.e.* an extracellular alkalosis which is aggravated by the loss of potassium from the cells, to which further reference will be made. The main clinical features of alkalosis are phasic respiration and latent tetany. Apnoeic periods of *up to half a minute's* duration are followed by similar periods of normal breathing. Chvostek's facial nerve and Trousseau's

main d accoucheur tests may reveal the presence of latent tetany Frank convulsions and air hunger only occur when there is a very severe degree of alkalosis such as may result from the continued ingestion of soluble alkalis by a peptic ulcer patient with stenosis

SODIUM LOSS —The tonicity of body fluids is largely controlled by their *sodium* content and sodium is responsible for holding water in the body When gastric mucus and juice of low acidity are vomited considerable quantities of sodium are lost (Davies *et al* 1956) As a result more water is excreted by the kidneys and some water enters the cells For a time the plasma proteins succeed in maintaining the blood volume but eventually this mechanism fails haemoconcentration develops and leads to hypovolaemic circulatory collapse The kidneys do however conserve sodium in sodium depletion Since less of this base is being excreted the reaction of the urine may remain acid despite the presence of an alkalotic state

SODIUM DEFICIENCY —Loss of sodium (which always involves the loss of water) causes extracellular hypotonicity The normal elasticity and turgor of the skin and subcutaneous tissues are lost the tongue becomes shrunken and intraocular tension is lowered The blood pressure eventually falls and the pulse rate rises the veins are empty cyanosis may develop and renal failure become manifest

POTASSIUM LOSS —Any patient who has been vomiting for more than three or four days is likely to be suffering from a deficiency of *potassium* Such a deficiency was present in five out of the ten patients studied by Davies *et al* (1956) The deficiency is due to cessation of intake to the prolonged vomiting to continued excretion by the kidneys (which do not conserve potassium in the way they conserve sodium in depleted states) and to the adrenocortical response to illness which increases potassium loss The considerable reserves of potassium (ninety-eight per cent of the total body content) within the cells are drawn upon in deficiency but are neither readily nor rapidly available The great preponderance of intracellular potassium over circulating potassium renders plasma levels of relatively little significance The withdrawal of potassium from the cells affects many of their functions Some of the potassium ions may be replaced by hydrogen ions migrating into the cells the resulting diminution in hydrogen ion concentration in the extracellular tissues may cause alkalosis which is not correctable unless potassium is given (Black and Jepson 1954)

POTASSIUM DEFICIENCY —Potassium is essential for normal myoneural and cardiac activity When there is a deficiency of potassium the muscles become weak and hypotonic The patient may be found lying slumped in

bed in bizarre positions (Wilkinson 1955) and drowsiness and apathy are common Frequently the abdomen is distended in a manner similar to that found in paralytic ileus The veins may be unusually full and an electrocardiograph shows flattening or inversion of the T wave and prolongation of the Q T interval

Assessment of deficiencies and their correction

GENERAL PRINCIPLES —Although chloride deficiency is gross it may be ignored because it is automatically made good if sodium and potassium are replaced in the form of their chlorides

Day to day needs of water Na and K can and should be estimated from the amount lost in all vomit urine and from other sources It remains therefore to estimate the losses of water Na and K already sustained when treatment is first undertaken

It is not possible to measure any of these unknown losses by any simple *or single laboratory or other investigation*

Consequently frequent clinical examinations to assess the response to treatment and accurate fluid balance charts are of the greatest importance The patient s fluid and electrolyte requirements must be recalculated once or twice every day

With these principles in mind the attempt to estimate requirements is embarked upon

ESTIMATION OF FLUID AND ELECTROLYTE DEFICITS —*Water* The total fluid requirement is the sum of the estimated pre-existing or established losses the normal metabolic requirements and the observed losses

Some idea of the established fluid losses may be obtained from the observation that when slight signs of dehydration are present the fluid deficit is usually of the order of one and a half to three litres while with severe dehydration it may be up to five litres (Le Quesne 1957) When moderate dehydration is present the deficit equals six per cent of the body weight (one stone wt = 6 4 kg)

A more accurate estimate of fluid requirements on admission is given by the plasma specific gravity Empirically each 0 001 difference from the normal P S G of 1027 indicates a fluid deficit of 200 ml in an average male Thus in such a man a P S G reading of 1032 shows that 1 000 ml *of fluid is required rapidly*

Sodium chloride Sodium and chloride ions are usually considered together in terms of normal (0 9 per cent) saline because in replacement healthy kidneys can compensate for minor relative inaccuracies Sodium is the more urgent need The total *sodium deficit* in pyloric stenosis varies within wide

limits In moderate cases it may be 100 500 mEq while in severe cases over 1 000 mEq of Na may be needed Repeated serum electrolyte estimations provide evidence of the adequacy or otherwise of sodium replacement

Potassium The estimation of potassium deficit is mainly based on the clinical findings and E C G since the serum potassium concentrations may remain normal in spite of dangerous degrees of cellular depletion In pyloric stenosis of moderate degree the body deficit may be 50 200 mEq but in severe cases with obvious clinical and E C G evidence of potassium deficiency the deficit may be up to 800 mEq of potassium

GENERAL ASSESSMENT AND INVESTIGATIONS BEFORE AND DURING THERAPY —*Clinical assessment* The patient s condition should be re assessed at least four times during the first twenty four hours and thereafter he should be examined twice daily until all deficiencies have been fully corrected The pulse and respiratory rates and blood pressure are recorded at frequent intervals

Urinary output and specific gravity —These should be carefully charted An output of over 1 000 ml per day with S G 1015 or less is desirable fluid administration should be continued until figures of this order are obtained and show that fluid replacement is complete

Blood urea —An elevated blood urea level gives an indication of the degree of renal impairment resulting from sodium and water loss The blood level should be checked at intervals during treatment until it returns to normal

Plasma specific gravity and haematocrit —The plasma specific gravity can be quickly measured using standard copper sulphate solutions (Phillips *et al* 1950) The normal P S G is 1027 In men the normal haematocrit (packed cell volume) is forty seven per cent and in women is forty two per cent Either of these tests may reveal haemoconcentration which must be corrected by giving water and sodium

Serum Na K and bicarbonate (electrolyte block)—About 20 ml of blood should be carefully collected in a dry heparinised tube Serum levels of course only indicate concentrations not absolute amounts Haemoconcentration and ionic shifts may render these investigations unreliable as a guide to intracellular deficiencies and clinical assessment is frequently more accurate Serial electrolyte estimations show how the patient is responding to treatment but clinical improvement frequently precedes demonstrable biochemical improvement The fallibility of normal serum potassium readings has been sufficiently stressed already

ADMINISTRATION —There are limitations to the rates at which certain deficiencies may be made good

CHAPTER XI

ALIMENTARY FUNCTION FOLLOWING GASTRIC OPERATIONS

By RICHARD B. WELBOURN

RESERVOIR FUNCTION OF THE STOMACH

THE stomach prepares food for the small intestine by diluting, mixing, liquefying and partially digesting it. Elaborate nervous and humoral mechanisms control the rate of gastric emptying so that food reaches the duodenum at a suitable rate and in a proper condition for the major part of its digestion and subsequent absorption (Fenton and Cowgill, 1949).

Gastric motility

The motility of the stomach and the manner in which it empties have been studied by kymography and by radiography. The rate at which the stomach empties has been measured after the addition of food to a standard barium meal (Shay and Gershon-Cohen, 1935; Meurling, 1953; Pulvertaft, 1953).

Operations on the stomach modify its capacity and rate of emptying. *Gastro-enterostomy* does not reduce the capacity, but usually causes a slight increase in the rate of emptying. Sometimes the increase is great (Springs and Marxer, 1922). Occasionally mechanical faults or scarring at the stoma prolong the emptying time. *Partial gastrectomy* reduces the capacity and increases the rate of emptying in direct proportion to the extent of the resection (Visick, 1948), while total gastrectomy allows the whole meal to pass straight into the small bowel in an unprepared state. The partially resected stomach empties more slowly when it is anastomosed to the duodenum (Billroth I*) than when the duodenum is by-passed (Polya*) (Butler and Capper, 1951).

The gastric remnant tends to increase in size during the first few months after resection. The enlargement can be recognised radiographically and by observing at a subsequent laparotomy the distances which separate the vasa brevia. The upper foot or so of the efferent jejunal loop often undergoes hypertrophy, especially after total gastrectomy.

After *vagotomy* the stomach empties very slowly. Kymographic studies, however, show that, while propulsive waves are greatly reduced, mixing waves

* The terms Billroth I and Polya are used loosely to describe gastrectomies with gastro-duodenal and gastro-jejunal anastomoses respectively.

are much increased (Hightower *et al* 1950) The propulsive waves can be largely restored by cholinergic drugs and tend to return spontaneously with the passage of time Gastric retention may cause troublesome symptoms Gastro-enterostomy pyloroplasty or gastrectomy performed at the same time as vagotomy allows the stomach to empty more readily

Effects of rapid passage of food into the intestine

FLUID AND CIRCULATORY DISTURBANCES —Many foods especially those which contain sugar or which can be hydrolysed rapidly into sugars or amino acids form hypertonic solutions in the lumen of the bowel An intact stomach allows these solutions to reach the small intestine at an appropriate rate If they pass quickly into the jejunum water is rapidly attracted into its lumen (Machella 1950) with consequent reduction of the volume of the blood plasma (Roberts *et al* 1954 Duthie *et al* 1959) This in turn may cause circulatory dis turbances which include changes in the heart rate blood pressure and E C G and a decrease in cerebral blood flow (Auguste 1954) These changes may be partly responsible for the early post-cibal dumping syndrome There is no increase in the total splanchnic blood flow (Ihre 1958 personal communica tion)

ALTERED ALIMENTARY MOTILITY —A rapid increase in bulk of the contents of the bowel provides a strong stimulus to peristalsis After gastrectomy a standard barium meal usually passes more rapidly than normal through the small intestine (Hartfall 1934 Welbourn 1953) and a meal of barium mixed with hypertonic glucose may reach the colon within a few minutes (Glazebrook and Welbourn 1952) The transit time from mouth to anus is reduced particularly after total gastrectomy Hypermotility sometimes causes symp- toms and may interfere with digestion and absorption of food

Occasionally the motility of the bowel is *reduced* after gastrectomy especially when it is combined with vagotomy and abnormal motility patterns may be recognised (Glazebrook 1952 MacPhee 1953) The effects of vagotomy alone in man are uncertain but clinical evidence of abnormal motility is provided by the occasional development of colic and diarrhoea and by the rare occurrence of intussusception (Grimson *et al* 1950)

ABSORPTION OF SUGAR —Glucose is absorbed very rapidly from the jejunum and after gastrectomy or gastro-enterostomy the blood sugar often reaches a very high level shortly after a meal This causes no ill effects but may result in alimentary glycosuria An erroneous diagnosis of diabetes is sometimes made especially when there is associated loss of weight An abnormally *low* blood sugar level frequently follows within one and a half

to two hours and this is said to be the result of reactive hyperinsulinism (Smith *et al* 1953) It may give rise to symptoms of hypoglycaemia Post cibal blood sugar curves of this type are found occasionally in otherwise healthy subjects and more frequently in those with duodenal ulcer (Butler 1951) They are found in most patients after gastrectomy and in many after gastro-enterostomy Vagotomy for some reason appears to accentuate the abnormality (Hastings James 1949 Butler 1951)

A slight but definite fall in the serum level of potassium accompanies the rapid deposition of glycogen in the tissues after the hypoglycaemic phase (Smith 1951) but this is probably of no importance (Munck 1954 Roberts *et al* 1954)

Alcohol is probably absorbed more rapidly after gastrectomy than before since some patients notice a reduction in their tolerance to it

DIGESTION AND ABSORPTION OF FOOD —In contrast with the rapid and complete absorption of carbohydrates into the blood stream the digestion and absorption of fat and protein are often impaired The impairment contributes to a varying degree to the loss of weight which is so common after operations on the stomach It has been known for many years that there are undigested food residues and an excess of fat in the faeces (De Filippi 1894a and b Wroblewski 1898 Gordon Taylor *et al* 1929) More recently many careful quantitative studies have been made of the intake absorption and excretion of fat and of nitrogen after different types of operation in man and in animals (Emery 1935 Wollaeger *et al* 1946 Brain and Stammers 1951 Everson 1952a and b Welbourn *et al* 1953 Naish *et al* 1954 Shingleton *et al* 1956 1957 Johnston and Welbourn 1958) The terms steatorrhoea and creatorrhoea indicate respectively an increase of fat or of nitrogen in the faeces

After gastrectomy fat (labelled with radioactive iodine) appears in the blood stream earlier than normal following a meal but the maximum concentration is lower than normal The proportions of split and unsplit fat in the faeces are unaltered The excretion of fat increases in proportion to the extent of the gastric resection Normally less than five per cent of the ingested fat is excreted After gastro-enterostomy excretion is usually normal while after total gastrectomy half the ingested fat may be found in the faeces After partial resection a Billroth I anastomosis causes less disturbance than a Polya Vagotomy does not usually cause steatorrhoea but may do so in some patients with symptoms of hyperactivity of the small bowel (Fox *et al* 1950) Steatorrhoea often diminishes with the passage of time but sometimes becomes worse

The digestion and absorption of protein is impaired less than that of fat and significant creatorrhoea is usually found only after total gastrectomy

The cause of the increased excretion of food constituents is not known precisely Most of the fat and nitrogen are probably derived directly from food but it is possible that some may be synthesised by bacteria or excreted by the intestinal mucosa Factors which are probably important are inadequate mixing of food and digestive juices (Lundh 1958) absence of gastric juice (Johnston and Welbourn 1958) decreased pancreatic secretion (Annis and Hallenbeck 1952) and intestinal hurry Growth of bacteria in the small intestine (Frazer 1949) or in an intestinal cul de sac (Naish and Capper 1953) may possibly play a part

The absorption of iron from food is impaired after gastrectomy with the result that anaemia often develops Radioactive tracer studies indicate that inorganic iron (Smith and Mallett 1957) and organic iron incorporated in a light meal are absorbed normally However when organic iron is given with a full meal that is in the form in which patients normally obtain it the absorption is reduced (Baird *et al* 1959) The cause of this defect is not yet clear but it is probably related to the by passing of the duodenum (in the Polya operation) and to the rapid passage of unprepared food through the upper jejunum Achlorhydria may also play a part it is probably important in the rat (Welbourn and Doggart 1956) but not in the dog (Johnston and Welbourn 1958) its role in man is not known

SECRETORY FUNCTION OF THE STOMACH

Hydrochloric acid and Pepsin

Abolition of the peptic activity or digestive power of the gastric juice is a major objective in the surgery of peptic ulcer Peptic activity is maximal at a *p*H between 1 and 3 less than twenty per cent of the maximum at *p*H 4 and absent at a *p*H of 5 or more (Hollander 1946) If free acid is present in gastric juice the *p*H is 4 or less It may therefore be assumed that if no free acid is found in the stomach during rest or after stimulation the operation has been effective in this respect

Gastric acidity is not altered appreciably by gastro enterostomy (Holman and Sandusky 1938) and after limited partial resections acid is still secreted freely (Gordon Taylor *et al* 1929 Strauss *et al* 1937) Subtotal Polya resection in which all the body and the pyloric antrum (which secretes gastrin) are removed is followed by histamine fast achlorhydria in a high proportion of cases (Wangensteen and Lannin 1942 Watson 1947) The fact that no free acid is found in a test meal after gastrectomy does not necessarily mean that no acid is being secreted It may simply indicate that the stomach is emptying rapidly and that bile and pancreatic juice are neutralising the acid

as fast as it is formed A few patients who develop jejunal ulcers after sub-total Polya resection secrete more acid than normal subjects with intact stomachs (Marks 1957) Free acid is found more often after Billroth I than after Polya resections (Morley and Bentley 1938) even when the resection has been high (Welbourn unpublished data) Radical segmental gastric resection with removal of ninety per cent of the acid bearing area of the stomach anastomosis of the antrum to the fundus and pyloroplasty (Wangensteen 1952 produces histamine fast achlorhydria in over ninety per cent of cases (Maclean *et al* 1953) After total gastrectomy no acid is secreted Complete vagotomy either alone or with resection of one third of the stomach at its pyloric end reduces the acidity of the gastric contents particularly in the fasting state, but free acid is still usually found Vagotomy combined with fifty per cent resection appears to control acidity as effectively as sub-total resection alone (Farmer *et al* 1951) Hypoglycaemia induced by insulin normally stimulates the medulla to provide vagal impulses which cause the stomach to secrete acid Complete vagotomy abolishes this secretion but incomplete nerve section does not (Hollander 1946)

The reaction in the whole of the upper alimentary tract is usually about neutral (*p*H 7) after sub total or total gastrectomy (Milanes *et al* 1944 Brain and Stammers 1951 Welbourn 1953) although it may be frankly acid below the stoma of a gastro enterostomy or limited partial gastrectomy (Panthitschko and Schmid 1950)

The anacidity of the upper part of the alimentary tract may interfere with the absorption of food (Johnston and Welbourn 1958) organic iron (Baird *et al* 1959) and vitamins may reduce the external secretion of the pancreas (Annis and Hallenbeck 1952) and may alter the alimentary flora Aerobic coliform bacteria may be cultured from the stomach contents of about fifty per cent of patients who have undergone sub-total gastrectomy but not from normal subjects (Welbourn 1953) These organisms are probably transient and do not colonise the upper small intestine (Duncan *et al* 1954) but they may possibly interfere with the absorption of fat and the B vitamins Anaerobic organisms such as Cl welchii do however grow in the gastric remnant and produce detectable amounts of α toxin This may sometimes cause diarrhoea (Howie *et al* 1953)

The bacteriology of the stomach after simple vagotomy does not appear to have been studied but clinical evidence suggests that the gastric delay and hypo-acidity cause fermentation of the retained food

Specific factors

The intrinsic factor of Castle is produced by the body and to a smaller extent by the fundus of the stomach (Fox and Castle 1942) but there is no

direct evidence of an extra gastric source in man Sub total gastrectomy removes most of the mucosa which elaborates the factor while total gastrectomy removes it all (Paulson *et al* 1950 Callendar *et al* 1954) The absorption of vitamin B_{12} is reduced by sub-total gastrectomy and abolished by total gastrectomy (Glass *et al* 1955) Consequently after total gastrectomy the body s store of vitamin B_{12} and its concentration in the serum fall progressively and megaloblastic changes develop in the bone marrow (Girdwood 1956) Absorption is restored to normal by the feeding of intrinsic factor The secretion of gastric muco protein (which is related to the intrinsic factor) is apparently not influenced by partial gastrectomy although it is reduced by vagotomy (Mersheimer *et al* 1952) The change in reaction of the stomach contents is unlikely to affect the interaction of intrinsic and extrinsic factors for this occurs optimally at a *p*H of 7 (Castle *et al* 1937)

There is evidence that a substance (possibly glucagon) which raises the blood sugar is produced by the stomach and that its activity is reduced by gastrectomy (Friedman *et al* 1955 Ivic and Kanesic 1956) It has been claimed that the removal of other specific factors found in gastric juice affects the body in various ways after gastrectomy but the evidence is incomplete

FUNCTION OF THE GALL BLADDER, PANCREAS AND AFFERENT JEJUNAL LOOP

The gall bladder usually enlarges progressively after gastrectomy or vagotomy and doubles its volume within a year of operation The probable cause of the dilatation is interruption of the parasympathetic nerve supply The rate of emptying of the gall bladder in response to a fatty meal is increased probably as a result of the rapid entry of food into the intestine (Cox *et al* 1958)

The volume of pancreatic juice which is secreted in response to a meal is reduced by Polya gastrectomy but can be restored by the introduction of hydrochloric acid into the duodenum or by the intravenous injection of secretin (Annis and Hallenbeck 1952 MacLean *et al* 1954)

Normally bile and pancreatic juices are mixed with the liquid chyme as it passes through the duodenum Polya gastrectomy and to some extent gastro enterostomy convert the duodenum and afferent jejunal loop functionally into an extension of the bile and pancreatic ducts When unaltered food is emptied rapidly into the efferent loop the juices tend to follow it down the intestine instead of mixing with it first (Lundh 1958) Sometimes there is delayed emptying of the afferent loop which may cause vomiting of bile and other symptoms after eating and probably interferes with the digestion and absorption of food Rarely the afferent loop empties so poorly that its contents

are permanently stagnant and support bacterial growth The stasis may cause various intestinal cul-de-sac phenomena including severe malnutrition (Nash and Capper 1953)

When the jejunal anastomosis is made from left to right (Moynihan) food usually passes into the afferent instead of the efferent loop and mixes rapidly with the digestive juices Later the mixture is expelled and passes smoothly down the small intestine (Pannett 1950) Disturbance of this mechanism may cause symptoms

STRUCTURAL MUCOSAL CHANGES

Structural changes have been observed in the mucosa of the stomach by gastroscopy and by biopsy (Palmer 1954 Schindler and DiGrndi 1955 Joske *et al* 1955) and in the mucosa of the jejunum by biopsy (Baird and Dodge 1957) They are not obviously related to any clinical syndrome or functional disorder and their significance is unknown During the first two to four weeks after operation there is acute generalised gastritis with oedema hyperaemia and some purulent exudate At all later periods areas of necrobiosis and acute inflammation are found in the superficial mucosal layers especially near the stoma in over fifty per cent of patients These changes are probably caused by intestinal juice entering the stomach Vagotomy appears to afford some protection to the stomach although it has been observed to cause mucosal atrophy in the dog (Rudik 1952) In the intact stomach similar lesions often precede general mucosal atrophy but in the operated stomach they are not progressive (Palmer 1954) Rodgers (1958 personal communication) however is impressed by the frequency with which an atrophic mucosa is seen gastroscopically some years after gastro-enterostomy and points out that this change is rare pre operatively in patients with duodenal ulcer Hypertrophy of the gastric mucosa has been reported by some gastroscopists (Bruusgaard 1946 Schindler and DiGrndi 1955) but has not been confirmed by biopsy Changes in the jejunal mucosa which are found in about one third of patients include hyperaemia oedema and mild atrophy of the villi and mild inflammation at the stoma

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CHAPTER XII

THE DELAYED COMPLICATIONS OF OPERATIONS ON THE STOMACH

By RICHARD B WELBOURN

THE results of modern operations for the relief of peptic ulceration are among the best in surgery Permanent relief of ulceration freedom from serious side effects and ability to work and enjoy life are achieved in about ninety per cent of cases (Pulvertaft 1952 Mercer 1954) Operative deaths and recurrent ulceration account for less than half of the ten per cent of failures while other serious side effects of gastric surgery account for the rest Many of the ninety per cent suffer minor degrees of disability or are nutritionally subnormal but they are unquestionably better than they were before operation and very grateful for the relief which they have obtained These few serious and many minor disturbances are often called the post gastrectomy syndromes (Wells and Welbourn 1951) The term is convenient but not strictly accurate since they may follow operations such as gastroenterostomy or vagotomy in which no stomach is resected Their reported frequency and severity vary enormously and it is clear that those who have found a low incidence have either examined their patients superficially or have included only those who complained spontaneously Those who have found these syndromes in a high proportion of cases have included all those patients from whom minor symptoms have been elicited on close questioning or in whom slight nutritional deficiencies have been found

The purpose of this section is to describe these undesirable effects of gastric surgery to discuss their aetiology and pathogenesis and to indicate how they may be treated

Classification

Five main groups of syndromes (apart from those which have been described elsewhere p 126) may develop after operations on the stomach They will be considered in turn

Early post-cibal syndromes*

Abdominal fullness

The dumping syndrome

Bilious vomiting

* The term post cibal is preferred to post prandial for etymological reasons in Latin cibus means food in general while prandium means a late breakfast or lunch (Lewis and Short 1951)

Hypoglycaemic (late post-cibal) syndrome

Other alimentary symptoms

Vomiting of food

Anorexia and nausea

Colic diarrhoea constipation and steatorrhoea

Disturbances of nutrition

Loss of weight

Iron deficiency anaemia

Megaloblastic anaemia

Vitamin B complex deficiency states

Severe malnutrition

Pulmonary tuberculosis carcinoma of the stomach and coronary thrombosis

Four points must be remembered in approaching the problem of ill health following gastric surgery First it must not be assumed that it is necessarily caused by the operation Other lesions such as hiatus hernia carcinoma or tuberculosis may cause similar symptoms and must be excluded Secondly symptoms such as those of the dumping syndrome hypoglycaemia and anaemia are sometimes present *before* operation and should be recognised and taken into account when surgery is considered Thirdly while abdominal pain may indicate recurrent ulceration (which is now uncommon) it does not necessarily do so Lastly two or more syndromes are often present at the same time in one individual

EARLY POST CIBAL SYNDROMES

This term is used to distinguish symptoms which are experienced immediately after eating from the late post cibal or hypoglycaemic symptoms which do not develop until later (Fig 35) Although the syndromes have many features in common they must be clearly distinguished (Adlersberg and Hammerschlag 1947)

Clinical features

ABDOMINAL FULLNESS —Almost every patient who has undergone gastrectomy finds that his capacity for food is greatly reduced immediately after operation The commonest limiting factor is a feeling of fullness pressure or distension in the epigastric and umbilical regions identical with that which a normal subject feels after an unusually large meal After gastrectomy however it may develop after only a few mouthfuls and it may be so severe that the patient describes it as a pain In most cases the capacity for food steadily increases until after a few months three good meals may

be eaten in the day but three-quarters of the patients who have undergone sub-total gastrectomy never quite regain their normal capacity (Welbourn 1953) This symptom is a major cause of loss of weight after gastrectomy but causes no serious disability unless the patient undertakes heavy manual work

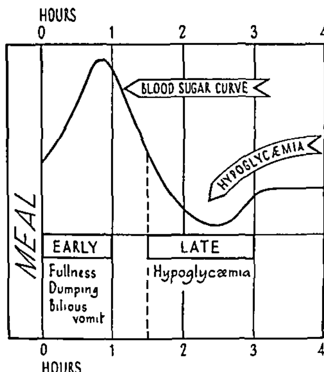


FIG 35
Time relations of post-cibal syndromes Early and late syndromes are shown in relation to a meal and to the blood sugar curve (after Wells and Welbourn 1951)

THE DUMPING SYNDROME—This term describes a symptom-complex which develops at some time after gastrectomy in at least one third of cases but which is severe and lasting in very few It occasionally follows gastro enterostomy The abdominal fullness which has been described already is associated with

1 Drowsiness fatigue and muscular weakness
and/or

2 Vasomotor symptoms such as palpitations sweating and a feeling of warmth

These features are pathognomonic Nausea and symptoms of alimentary hypermotility such as colic borborygmi and diarrhoea may be present but alone they do not constitute the syndrome (Capper and Welbourn 1955)

Symptoms develop during or within a few minutes of finishing a meal and last from a few minutes to two or three hours in severe cases They may compel the patient to lie down and when he does so he obtains relief

While the symptoms are present the patient often appears pale The heart rate and blood pressure are usually increased but occasionally (perhaps in the more severe cases) they are unchanged or even reduced (Meurling 1953)

Symptoms are usually noticed when the first full meal is eaten after operation and are maximal in the first few weeks or months They then improve steadily and half the patients lose them completely within eighteen months (Mercer 1954) Thereafter they change little although occasional cases are met in which they develop for the first time years after operation A few people with intact stomachs experience similar symptoms

Various dietary factors influence the development of symptoms (Meurling 1953) The most important are

1 The *size* of the meal which the patient will often limit to avoid symptoms

2 The *consistency* of the meal dry or solid foods being tolerated better than fluids

3 The *nature* of the meal sweet and milky foods being particularly likely to cause symptoms Occasionally patients appear to have an idiosyncrasy for one particular food such as eggs

BILIOUS VOMITING —This symptom develops at some time after operation in about one quarter of patients following gastrectomy or vagotomy and gastro enterostomy and occasionally after gastro enterostomy alone Shortly after a meal clear bile stained fluid (often described as acid) unmixed with food is regurgitated or actually vomited This occurs at intervals varying from several times a day to once in many months The quantity varies from a mouthful to a cupful It is often accompanied by a burning sensation in the throat and oesophagus and a bitter taste in the mouth and sometimes the patient notices these symptoms without being aware of the regurgitation of fluid Abdominal fullness often localised in the right hypochondrium nausea or dumping symptoms may precede the bilious vomiting and may be relieved or ameliorated by it

Occasionally the symptoms are noticed at other times for example on waking and are not strictly post-cibal Rarely at intervals of a few months abdominal symptoms steadily increase for a day or longer and the motions become pale bulky and offensive At the end of this period a large quantity (often described as one or two pints) of clear bile is vomited with immediate relief of the symptoms

Like the dumping syndrome bilious vomiting usually diminishes steadily for the first year or so after operation and disappears completely in about one third of cases (Mercer 1954) This syndrome however is more likely to recur or to develop for the first time in later years

The size consistency and nature of the meal are similarly important and cooked fats are particularly likely to cause symptoms (Mercer 1954)

Two conditions with which this syndrome may be confused or associated particularly when the symptoms are atypical are hiatus hernia and chronic cholecystitis Radiography however should resolve any doubt

Aetiological factors

Several factors influence the development of the early post-cibal symptoms (Capper and Welbourn 1955) Some depend on the clinical and pathological state and others on the nature of the operative procedure

CLINICAL AND PATHOLOGICAL

Sex —Although mild symptoms are equally common in the two sexes women suffer severe symptoms twice as commonly as men

Severity of the lesion —Those with severe lesions (stenosis penetration multiple or large ulcers etc) have far fewer complaints afterwards than the others but there is no evidence that those with scars or small ulcers only do any worse than those with average lesions

Allergy —Those who have suffered from allergic diseases are particularly liable to develop dumping symptoms (Meurling 1953)

Anaemia and sideropenia —Dumping symptoms are said to be most frequent in those who are anaemic and have low concentrations of iron in the serum (Wallensten 1955) This however is not the general experience (Remy *et al* 1953)

The age and length of history do not affect the result Many surgeons believe that those with mild symptoms before operation or those who have symptoms out of proportion to the severity of their lesions make much of their disabilities afterwards but these points have not been adequately investi gated

NATURE OF THE OPERATIVE PROCEDURE

Extent of resection —Gastro-enterostomy (in the absence of recurrent ulceration) is rarely followed by symptoms of any sort and the clinical result is usually excellent Post-cibal symptoms however are common after nearly

all types of gastrectomy There is little clear evidence that either dumping symptoms of bilious vomiting are influenced by the extent of the resection although many believe that they are more troublesome following high sub-total gastrectomies than they are after more limited resections (Wells and MacPhee 1952)

TABLE VI

COMPARISON OF RESULTS OF GASTRECTOMY WITH GASTRO DUODENAL (BILLROTH I) AND GASTRO-JEJUNAL (POLYA) ANASTOMOSES

(Data from Capper and Welbourn 1955)

Undesirable effects	*Gastro duodenal anastomosis*	*Gastro jejunal anastomosis*
Dumping syndrome		
Moderate and severe*	4 0	8 0
Mild	15 4	15 9%
Bilious vomiting		
Severe*	3 0 %	9 6
Mild*	3 5	13 7%
Colic diarrhoea and steatorrhoea	Less	More
Loss of weight	Less	More
Anaemia	Less	More

* These differences between the results of the two anastomoses are statistically highly significant
(P<0 01)

Gastro duodenal (Billroth I) or gastro-jejunal (Polya) anastomosis (Table VI) Following partial gastrectomy a Billroth I* anastomosis gives a better functional result than one of the Polya* type (Table VI) The most striking difference is in the incidence of bilious vomiting which is far commoner and much more severe following the latter Dumping symptoms also are more often severe and colic and diarrhoea are more frequent after a Polya anastomosis Against these advantages of the *Billroth I procedure must* be set the disadvantage of a higher recurrence rate when the operation is performed for duodenal ulcer (p 220) The results of operations in which segments of colon (Moroney 1953) or of jejunum (Henley 1953) are inserted between the gastric remnant and the duodenum may be superior to those which follow gastro-jejunal anastomoses but are not obviously better than those of a direct gastro-duodenal anastomosis (and are probably more liable to recurrent ulcera

* The terms Billroth I and Polya are used loosely to describe gastrectomies with gastro duodenal and gastro jejunal anastomoses respectively

tion) Radical segmental gastric resection (Wangensteen 1952) is no better than the Polya operation (MacLean *et al* 1953)

Types of gastro-jejunal anastomosis —Most of the arguments about the relative merits of different types of anastomosis (ante-colic or retro-colic right to-left or left to-right small or wide stoma with or without a valve) are theoretical and there is little difference between the results obtained with them However a Roux-en Y anastomosis reduces bilious vomiting (Schofield and Anderson *1953* *Hall 1954*) *Its* effect on dumping symptoms is un certain for some (Hall 1954) have encountered them while others (Schofield and Anderson 1953) have not Following total gastrectomy (Richardson and Jennings 1955 Brintnall *et al* 1956 Balint and Gummer 1958) and partial gastrectomy for hiatus hernia (Wells and Johnston 1955) it prevents the development of oesophagitis from biliary regurgitation

Vagotomy —Early post-cibal symptoms are probably as common after vagotomy and gastro-enterostomy as they are after gastrectomy (Jordan *et al* 1952 Everson *et al* 1957a Smart and Williams 1958) The late results of vagotomy and pyloroplasty are awaited with interest Vagotomy combined with a *limited* gastric resection particularly of the Billroth I type probably causes less trouble than a high resection alone (Farmer *et al* 1951 Johnson and Orr 1954 Moloney 1954)

Pathogenesis

It is convenient to consider the early post-cibal syndromes together since they are closely related Their pathogenesis is by no means clear and although they have been studied intensively there is little agreement among different investigators about the mechanisms involved

EXPERIMENTAL PRODUCTION OF SYMPTOMS —Experimentally several stimuli will reproduce the main features of the dumping syndrome in suscept ible persons If the stimulus is great enough symptoms may be produced in those who normally have none

1 Inflation of a balloon in the jejunum causes a feeling of fullness or distension in the centre of the abdomen (Butler and Capper 1951) often associated with nausea In a few patients it causes dumping symptoms (Machella 1950 Glazebrook and Welbourn 1952)

2 The introduction of hypertonic solutions especially of glucose into the lumen of the small intestine frequently reproduces all the features of the dumping syndrome often in a severe form and sometimes it causes bilious vomiting also (Machella 1950 Glazebrook and Welbourn 1952 Roberts *et al* 1954)

Attempts have been made to recognise before operation patients who will develop dumping symptoms afterwards by introducing hypertonic solutions into the intestine through a duodenal tube (Capper 1954 personal communication Fisher *et al*, 1955)

3 Rarely dumping symptoms are caused by drinking ice-cold water (Alvarez 1949) a large volume of iso-tonic fluid or a simple barium meal (Butler and Capper 1951)

4 Stretching of the gastric remnant after gastrectomy with a mercury weighted bag frequently causes dumping symptoms (Butler and Capper 1951)

PHENOMENA ASSOCIATED WITH DUMPING SYMPTOMS —Various phenomena which can be measured objectively may be associated with dumping symptoms It is not yet clear which of them if any are essential features of the syndrome

1 Changes in the heart rate and blood pressure are inconstant In an average attack they are probably raised and in a more severe one they may fall (Meurling 1953) The respiratory rate may also be increased (Smith *et al* 1953)

2 Various changes are found in the peripheral circulation Its volume is reduced as a result of the passage of fluid into the bowel It is not certain that the reduction is greater in those with symptoms than in those without some (Roberts *et al* 1954 Le Quesne 1957) have found that it is while others (Duthie *et al* 1959) believe that it is not There is peripheral vasoconstriction (Smith *et al* 1953) and reduction of visual auditory and vestibular acuity (Auguste 1954)

3 The E C G shows changes consisting of a lowering of the T wave increase of the P and U waves and depression of the S T segment (Smith *et al* 1953) These do not develop when glucose is given intravenously (Brotmacher 1954) but are common when food or various fluids are given by mouth They probably differ only in degree from changes which sometimes develop under similar conditions in those with intact stomachs (Gardberg 1954) Their cause and significance are not clear

4 The stomach remnant tends to empty more rapidly in those with dumping symptoms than in those without (Smith *et al* 1953 Meurling 1953 Pulvertaft 1953) and the distance which the gastric stoma descends (measured radiographically) on changing from a lying to an upright position is greater in those with symptoms than in those without (Butler and Capper 1951 Mercer 1954)

5 Hypermotility of the small intestine can be demonstrated radiographically and kymographically when dumping symptoms are induced by hyper tonic solutions Abnormal dilatation of the bowel does not occur (Glazebrook and Welbourn 1952)

EXPERIMENTAL RELIEF OF SYMPTOMS.—Dumping symptoms can be prevented or minimised by the following procedures

1 Lying down during or immediately after a meal This is about the only observation on which there is general agreement Bilious vomiting can often be avoided by lying down for a few minutes *before* a meal (Capper and Butler 1951)

2 Local anaesthetics taken orally or injected intramuscularly (Meurling 1953)

3 Bilateral paravertebral block of the splanchnic nerves and lumbar sympathetic chains which is effective in a high proportion of cases (Butler and Capper 1951) Right sided block may relieve bilious vomiting (Roux *et al* 1950)

4 Ganglion blocking agents such as hexamethonium bromide which are more effective parenterally than by mouth (Glazebrook and Welbourn 1952)

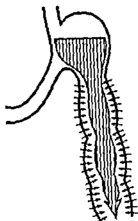
POSSIBLE MECHANISMS (Fig 36).—There is no one hypothesis which will explain all these observations Several are attractive but all have serious objections It seems reasonable however to draw the following conclusions

Abdominal fullness is a visceral sensation caused by rapid filling of the stomach duodenum or jejunum by food secretions and osmotically attracted water Sensations from the foregut are localised in the epigastrium and lower chest and from the midgut in the umbilical region A nervous pathway which starts in the sensory nerve endings in the bowel wall or mesentery and passes *via* the sympathetic nerves to the C N S is probably involved

The vasomotor phenomena—palpitations sweating pallor and alterations in the pulse rate and blood pressure—suggest widespread reflex activity of the autonomic nervous system initiated perhaps by stretching of the small bowel It has been suggested that *small bowel distension* is the effective stimulus (Machella 1950) but as has been pointed out already unless there is obstruction the bowel responds to stretching of its wall by contracting actively and not by distending This hypermotility may further stimulate sensory nerves and often causes other symptoms (borborygmi colic and diarrhoea) directly

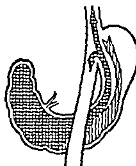
Other mechanisms however may be involved and may sometimes be the major factors First the mobility of the gastric stoma in those with symptoms suggests that traction by a loaded stomach and afferent jejunal loop may stimulate nerve endings in the region of the oesophago-gastric junction (Butler and Capper 1951) Another explanation however is that the [illegible] of the stoma allows the stomach to empty more rapidly [illegible] Second there may be chemoreceptors in the bowel wall which are [illegible]

I DUMPING SYNDROME



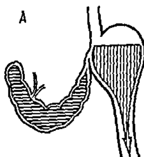
1 Rapid emptying of stomach
2 Osmotic attraction of fluid into lumen
3 Stretching and hypermotility of efferent loop

III AFFERENT LOOP REFLUX

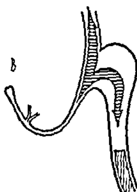


1 Moynihan anastomosis
2 Passage of food into afferent loop where it mixes with secretions
3 Distension and hypermotility
4 Sudden evacuation into stomach of food + secretions

II BILIOUS VOMITING



1 Passage of food down efferent loop
2 Functional obstruction of afferent loop
3 Accumulation of bile and pancreatic juice
4 Stretching and hypermotility of afferent loop



Sudden release of fluid into stomach

FIG 36

Probable mechanisms of early post-cibal syndromes (dumping syndrome and bilious vomiting) and of vomiting of food mixed with bile (afferent loop reflux)

to hypertonic solutions or specific substances Third a reduction in the circulating blood volume may initiate reflexes *via* the pressor receptors which control the blood pressure (Roberts *et al* 1954) and lastly a fourth possible mechanism is that pressor substances may be produced by the gut wall or reflexly by the supra renal medulla (Smith *et al* 1953)

The fatigue and weakness of the dumping syndrome are difficult to explain A reduced cerebral blood flow which has been postulated (Auguste 1954) but not demonstrated might cause them The main difficulty in accepting this theory is the beneficial effect of paravertebral block this would be expected to increase symptoms by causing vasodilatation in the splanchnic area and the legs and a further decrease in cerebral flow

Hypokalaemia resulting from the binding of potassium in the cells during the deposition of glycogen has been held responsible for these symptoms and for the electro-cardiographic changes (Smith *et al* 1953) The fall in the serum potassium level however is slight and does not occur until after the symptoms have passed (Munck 1954 Roberts *et al* 1954)

Bilious vomiting is also difficult to understand It has been called the afferent loop syndrome (Wells and MacPhee 1952) because it develops so frequently after operations with a gastro-jejunal anastomosis (see p 243) The term is not entirely satisfactory however since it does follow (although less frequently) operations of the Billroth I type (Table VI)

After operations with a gastro-jejunal stoma (that is when an afferent loop is present) the following is a possible mechanism In the erect position secretions which enter the loop accumulate but cannot leave easily because of kinking at the gastro jejunal junction caused by their weight and the high attachment of the loop to the lesser curvature of the stomach In the lying position the loop empties freely and up to one pint of bile stained fluid may be aspirated from a previously empty stomach As secretions increase during a meal the afferent loop starts to contract actively causing symptoms Eventually the obstruction is overcome whereupon the secretions enter the stomach rapidly and are partly vomited At operation the afferent loop is often hypertrophied but no obstruction is visible because the patient is recumbent Two unrelated observations support this hypothesis First after gastrectomy barium is often seen on radioscopy to enter the afferent jejunal loop but never in those who exhibit this syndrome (Wells and MacPhee 1952) Secondly spontaneous perforation of the afferent jejunal loop has been reported (Smith *et al* 1953) There are however objections to this explanation X ray studies with Bili grafin show that the afferent loop empties normally (Scott and Whiteside 1956) and suggest that the bile may pool not in the afferent loop but in the stomach or efferent loop (Capper and Airth 1957) There is no apparent correlation between the presence or absence of bilious vomiting and the degree

of post operative distension of the gall bladder or the rate at which it empties (Scott and Whiteside 1956 Cox *et al* 1958)

The mechanism following a Billroth I anastomosis is still more obscure In one case observed radiographically the tail of the barium meal was seen to pass down and up leaving the stomach and then re entering it repeatedly It is possible that as bile passes from the common duct it floats on the heavier viscous food and is forced back into the stomach from which it is regurgitated It is possible that the extensive mobilisation of the greater curvature which may be required to facilitate the anastomosis interferes with the cardio-oesophageal angle and favours oesophageal reflux (Capper 1955 personal communication) Wells and Johnston (1956) have in fact drawn a distinction between bilious vomiting and effortless bilious regurgitation they suggest that the latter is associated with a pre-existing hiatus hernia or with cardio oesophageal retraction secondary to abdominal vagotomy

DISCUSSION —Several observations suggest that the dumping syndrome may result from afferent loop dysfunction even in the absence of bilious vomiting and that it may represent a forme fruste of the afferent loop syndrome (Wells and MacPhee 1952) Thus dumping symptoms are sometimes associated with and may be relieved by the vomiting of bile and they sometimes disappear following operations which allow the afferent loop to empty freely Aspiration of bile before a meal may give freedom from dumping symptoms afterwards and relief of symptoms may follow the development of obstructive jaundice (personal observation) On the other hand dumping symptoms sometimes develop in the absence of an afferent jejunal loop vomiting of bile does not always relieve them and conversion of a gastro-jejunal to a gastro duodenal anastomosis occasionally aggravates them (Meurling 1953) Thus it may be that afferent loop dysfunction often contributes to the development of dumping symptoms but it is not their only cause

There are several possible reasons for the low incidence of early post-cibal symptoms following a Billroth I anastomosis First the stomach tends to empty more slowly than it does with a Polya anastomosis second there is no afferent loop dysfunction and third there is less drag on the oesophago-gastric region

Lying down may relieve symptoms in various ways First the stomach empties more slowly second the afferent loop empties more freely third drag on the oesophago-gastric region is avoided and a fourth possible mechanism is that cerebral blood flow may be increased

Treatment of early post cibal symptoms

The first essential is to make an *accurate diagnosis* of the condition This is done best from a careful history radiography or other special investigations being used to exclude other lesions

SIMPLE MEASURES

The patient must be re assured that his ulcer has been cured The cause of his symptoms should be explained to him in simple terms and if he has undergone operation recently he should be told that he will improve steadily and probably lose his symptoms completely within a few months

Advice about food should be given Meals should be taken as dry as possible and drinks should be taken *between* meals The size of the meals should be limited to that which the patient can tolerate but meals should be taken frequently to ensure an adequate intake of food A careful analysis should be made of the dietary habits to find out which foods cause symptoms and the following should each be avoided for a period in turn specific foods which are suspected of being causative milk and milky foods sugar and fats (especially cooked) Often an intelligent patient has done this for himself already All too often however his practitioner has advised him to return to an ulcer diet composed largely of milk and this has aggravated the symp toms Meat cheese butter bread fish and potatoes can usually be taken freely Salads and vegetables are usually well tolerated but are better avoided because they are bulky and contain few calories

These simple measures are usually effective If they fail *lying down* for half an hour *before* the main meal of the day should be tried and if this fails the meal should be eaten lying down

DRUGS

Drugs taken *by mouth* are usually disappointing (Capper and Welbourn 1955) but the following taken before meals are occasionally effective

1 Procaine hydrochloride—30 ml of one per cent solution
2 Lignocaine hydrochloride as a viscous suspension (Xylocaine Viscous Duncan Flockhart)—10 to 15 ml of a two per cent sus pension
3 Hexamethonium bromide—250 mg
4 Propantheline bromide (Probanthine Searle)—15 to 30 mg
5 Piperidyl benzilate methobromide (Piptal Benger)—5 mg
6 Sodium bicarbonate—4 gm or one teaspoonful

Iron given parenterally is said to be effective against dumping symptoms if there is anaemia or sideropenia (Wallensten 1955) iron deficiency should certainly be corrected (page 208)

If the condition is serious a *paravertebral block* (20 ml of amethocaine 1 20 000) at the level of L1 on each side is well worth while It relieves symptoms temporarily in most cases and permanently in a few It should certainly be tried before further major surgery is undertaken

OPERATIVE TREATMENT

Surgical measures should not be considered unless symptoms are severe and disabling and have persisted for at least a year after the initial operation Simpler measures must have been tried and failed and other possible causes of the symptoms must have been excluded Marked loss of weight and steatorrhoea provide objective evidence of bowel dysfunction which is helpful in assessing the degree of disability

Following the Polya type of gastrectomy or vagotomy and gastro-enterostomy conversion to a Billroth I anastomosis (Perman 1947 and 1954 Bohmansson 1950 Capper and Welbourn 1955) is the procedure of choice This is usually highly effective in relieving bilious vomiting and often improves dumping symptoms colic and diarrhoea and nutrition The results are better in men than in women There is a tendency for stomal ulceration to develop in men when the original operation has been undertaken for duodenal ulcer and particularly when a limited partial resection has been performed It is wise in these circumstances to perform a vagotomy preferably at the same time as the conversion

The operation is shown diagrammatically in Figure 37 The jejunal loops are found first freed from adhesions and traced to the stomach and a careful search is made for signs of obstruction or ulceration It is convenient to perform the vagotomy through the hiatus at this stage The duodenal stump is next sought and mobilised The jejunum is detached from the stomach and closed transversely The gastric stoma is narrowed suitably at the lesser curvature end and anastomosed into the end of the duodenum into the front of its second part or into both (p 112)

This procedure is not usually difficult but sometimes the stomach and jejunal loops are firmly stuck under the costal margin and cannot be mobilised easily In these circumstances the afferent loop should be detached from the stomach and the efferent loop used for bridging the gap between the stomach and the duodenum (Henley 1953) (Fig 37) Another alternative if bilious vomiting is the chief complaint is to detach the afferent loop and to insert it into the efferent loop lower down in the Roux-en Y manner (Capper and Welbourn 1955 Wells and Johnston 1956) In either case a vagotomy must be performed as a precaution against jejunal ulceration This will have to be undertaken through the chest at a later date if the adhesions around the stomach render the vagi inaccessible from below

Severe post-cibal symptoms which follow or persist after conversion to a Billroth I anastomosis present a very difficult problem but fortunately they are rare If paravertebral block gives temporary relief splanchnicectomy

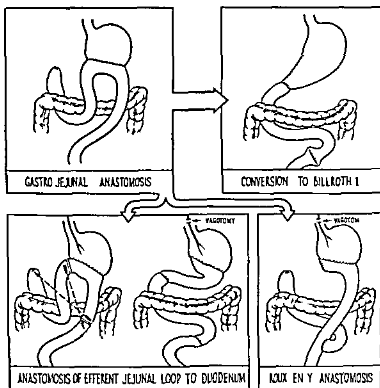


FIG 37
Operations for relief of early post cibal syndromes

might be worthy of trial Conversion to a Roux en Y anastomosis relieves bilious vomiting (Wells and Johnston 1955) The insertion of a colonic implant between gastric remnant and duodenum plus an abdominal vagotomy is being tried out to see if it will relieve symptoms by reducing the rate of gastric emptying (Wells 1959 personal communication) (p 119)

THE HYPOGLYCAEMIC (LATE POST CIBAL) SYNDROME

This syndrome develops in about five per cent of patients after all types of gastrectomy and also after vagotomy and gastro-enterostomy It was formerly confused with the early post-cibal dumping syndrome but it is distinguished by its occurrence at about one and a half to three hours after a meal (Fig 35) The patient often fails to relate the symptoms to his meals and leading questions are necessary to establish the diagnosis Rarely the early and the late syndromes develop in the same patient

The symptoms (Butler 1951) are those of hypoglycaemia the commonest being tremor giddiness sweating anxiety weakness hunger and an empty feeling in the abdomen There may also be nausea palpitations and headache and rarely loss of consciousness They are often precipitated by meals of high carbohydrate content and by exercise On examination during an attack the tremor and sweating are obvious and the patient is usually pale Tachycardia and a low blood pressure may be found There is often sugar in the urine and the blood sugar is usually less than 75 mg per 100 ml Symptoms are usually noticed for the first time a few weeks or months after operation they tend to diminish during the next two to three years but may persist intermittently Occasionally they do not develop until much later It is important to distinguish the condition clinically from diabetes especially if the patient loses consciousness and in medico legal cases it must not be confused with alcoholism

A diagnosis can usually be made on clinical grounds and by the immediate relief of symptoms by taking sugar When there is doubt a glucose tolerance test should be performed while the patient is ambulant so that the correlation between the symptoms and the blood sugar level can be tested

Symptoms are caused by the alimentary hypoglycaemia which usually follows gastrectomy or vagotomy The rate at which the stomach empties the absolute blood sugar levels and the plasma insulin concentrations are no different in those with symptoms than in those without The essential abnormality in the former group appears to be an over sensitivity to insulin (revealed by an intravenous insulin tolerance test) which causes the hypoglycaemia to persist longer than it does in the latter (Smith *et al* 1953) The sensitivity is not present immediately after operation but usually develops within a month (Barnes 1947 Butler 1951) Its cause is not known but it may possibly be related to the depletion of glycogen stores in the liver which accompanies post operative loss of weight It is interesting to note that the insulin requirements of diabetics may be reduced by gastrectomy (Friedman *et al* 1955)

Patients should be re assured and advised to eat snacks between meals and to carry lumps of sugar or sweets which can be eaten at once when attacks are imminent A diet containing plenty of protein and fat but little carbohydrate may prevent gross hyperglycaemia and so reduce the frequency and severity of hypoglycaemic attacks

OTHER ALIMENTARY SYMPTOMS

Vomiting of food

Vomiting of food following operations on the stomach is not nearly so common as vomiting of bile Often there is no obvious reason for it but

there are some recognisable causes Post-operative gastric retention has been discussed elsewhere (p 133)

Vagotomy alone regularly causes gross retention of gastric contents which undergo fermentation Abdominal fullness and epigastric colic often result and are relieved by belching of foul gas and by copious vomiting These symptoms may improve in time but persist in a severe form in at least one third of cases (Grimson *et al* 1950 Johnson and Orr 1954) Retention can be relieved or avoided by gastro-enterostomy pyloroplasty or gastrectomy and one of these procedures is now always carried out at the same time as vagotomy

Reflux of food into the afferent jejunal loop usually causes no trouble Occasionally however it causes dilatation of the duodenum (which may be gross) and symptoms such as fullness nausea or those of the dumping syndrome After a time the afferent loop empties itself forcibly into the stomach and food mixed with bile is vomited (Fig 36) Afferent loop reflux is easily demonstrated radiographically but need give rise to no anxiety unless it causes dilatation It occurs most commonly following gastrectomies with a left to-right (Moynihan) type of anastomosis and some have found it a common cause of disability after this type of operation (Mimpriss and Birt 1948) It is best treated by conversion to a Billroth I type of anastomosis

Mechanical disturbances in the jejunal loops such as intussusception and volvulus which are described elsewhere (p 136) are rare causes of vomiting of food

Anorexia and nausea

There are several vague and ill-defined symptoms which may develop after gastric operations which cannot be fitted easily into the recognised syndromes

Appetite is usually unimpaired even after total gastrectomy but some patients develop quite marked anorexia for no obvious reason Appetite can often be restored by a holiday or by treatment with iproniazid or methyl testosterone (p 207)

Nausea is common and may accompany dumping symptoms or precede any of the forms of vomiting which have been described Sometimes vomiting is induced to bring relief Nausea is sometimes the only symptom to which a patient will admit even on close questioning It is very hard to treat and further surgery is unlikely to help

Both these symptoms are more troublesome with high resections than with low and both require further study (Pulvertaft 1954 personal communication)

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Nausea is common and may accompany dumping symptoms or precede any of the forms of vomiting which have been described Sometimes vomiting is induced to bring relief Nausea is sometimes the only symptom to which a patient will admit even on close questioning It is very hard to treat and further surgery is unlikely to help

Both these symptoms are more troublesome with high resections than with low and both require further study (Pulvertaft 1954 personal communication)

The symptoms (Butler 1951) are those of hypoglycaemia the commonest being tremor giddiness sweating anxiety weakness hunger and an empty feeling in the abdomen There may also be nausea palpitations and headache and rarely loss of consciousness They are often precipitated by meals of high carbohydrate content and by exercise On examination during an attack the tremor and sweating are obvious and the patient is usually pale Tachycardia and a low blood pressure may be found There is often sugar in the urine and the blood sugar is usually less than 75 mg per 100 ml Symptoms are usually noticed for the first time a few weeks or months after operation they tend to diminish during the next two to three years but may persist intermittently Occasionally they do not develop until much later It is important to distinguish the condition clinically from diabetes especially if the patient loses consciousness and in medico legal cases it must not be confused with alcoholism

A diagnosis can usually be made on clinical grounds and by the immediate relief of symptoms by taking sugar When there is doubt a glucose tolerance test should be performed while the patient is ambulant so that the correlation between the symptoms and the blood sugar level can be tested

Symptoms are caused by the alimentary hypoglycaemia which usually follows gastrectomy or vagotomy The rate at which the stomach empties the absolute blood sugar levels and the plasma insulin concentrations are no different in those with symptoms than in those without The essential abnormality in the former group appears to be an over *sensitivity to insulin* (revealed by an intravenous insulin tolerance test) which causes the hypoglycaemia to persist longer than it does in the latter (Smith *et al* 1953) The sensitivity is not present immediately after operation but usually develops within a month (Barnes 1947 Butler 1951) Its cause is not known but it may possibly be related to the depletion of glycogen stores in the liver which accompanies post operative loss of weight It is interesting to note that the insulin requirements of diabetics may be reduced by gastrectomy (Friedman *et al* 1955)

Patients should be re assured and advised to eat snacks between meals and to carry lumps of sugar or sweets which can be eaten at once when attacks are imminent A diet containing plenty of protein and fat but little carbohydrate may prevent gross hyperglycaemia and so reduce the frequency and severity of hypoglycaemic attacks

OTHER ALIMENTARY SYMPTOMS

Vomiting of food

Vomiting of food following operations on the stomach is not nearly so common as vomiting of bile Often there is no obvious reason for it but

there are some recognisable causes Post-operative gastric retention has been discussed elsewhere (p 133)

Vagotomy alone regularly causes gross retention of gastric contents which undergo fermentation Abdominal fullness and epigastric colic often result and are relieved by belching of foul gas and by copious vomiting These symptoms may improve in time but persist in a severe form in at least one third of cases (Grimson *et al* 1950 Johnson and Orr 1954) Retention can be relieved or avoided by gastro-enterostomy pyloroplasty or gastrectomy and one of these procedures is now always carried out at the same time as vagotomy

Reflux of food into the afferent jejunal loop usually causes no trouble Occasionally however it causes dilatation of the duodenum (which may be gross) and symptoms such as fullness nausea or those of the dumping syndrome After a time the afferent loop empties itself forcibly into the stomach and food mixed with bile is vomited (Fig 36) Afferent loop reflux is easily demonstrated radiographically but need give rise to no anxiety unless it causes dilatation It occurs most commonly following gastrectomies with a left to-right (Moynihan) type of anastomosis and some have found it a common cause of disability after this type of operation (Mimpriss and Birt 1948) It is best treated by conversion to a Billroth I type of anastomosis

Mechanical disturbances in the jejunal loops such as intussusception and volvulus which are described elsewhere (p 136) are rare causes of vomiting of food

Anorexia and nausea

There are several vague and ill-defined symptoms which may develop after gastric operations which cannot be fitted easily into the recognised syndromes

Appetite is usually unimpaired even after total gastrectomy but some patients develop quite marked anorexia for no obvious reason Appetite can often be restored by a holiday or by treatment with iproniazid or methyl testosterone (p 207)

Nausea is common and may accompany dumping symptoms or precede any of the forms of vomiting which have been described Sometimes vomiting is induced to bring relief Nausea is sometimes the only symptom to which a patient will admit even on close questioning It is very hard to treat and further surgery is unlikely to help

Both these symptoms are more troublesome with high resections than with low and both require further study (Pulvertaft 1954 personal communication)

Diarrhoea, colic, constipation and steatorrhoea

Half the patients who *undergo gastrectomy* of any type have looser and more frequent bowel actions after operation than they had before. Less than five per cent however develop frank diarrhoea for the first time after operation although there are some who suffered from it before in whom it persists (Welbourn 1953)

Diarrhoea may follow every *meal* or *occur only at longer intervals*. *It* is often preceded by colic and borborygmi may accompany a severe attack of *dumping and is sometimes explosive in severity Undigested food* which was eaten at the previous meal is occasionally seen in the motions. A few patients who experience dumping symptoms lose them periodically and develop diarrhoea instead

Radiography often shows unusually rapid intestinal passage and kymography reveals excessive peristaltic activity (Glazebrook and Welbourn 1952) Rarely although the complaint is of diarrhoea X rays show a reduction of *motility with clumping of the barium (Glazebrook 1952 MacPhee 1953)*

These symptoms are much commoner following a Polya than a Billroth I anastomosis and are particularly troublesome after total gastrectomy and vagotomy They usually improve as time passes Their probable cause is the increased bowel motility which follows the rapid entry of food into the alimentary tract and they can often be relieved by ganglion blocking drugs such as propantheline bromide hexamethonium bromide and atropine In *some cases irritation by bacterial toxins produced in* the stomach after vagotomy or in a stagnant jejunal loop (Naish and Capper 1953) may be the major cause and *the diarrhoea can be controlled by chemotherapy and* antibiotics Dilute hydrochloric acid B P (up to 8 ml) taken during meals is sometimes effective although it is doubtful whether it has much effect on the *p*H of the bowel If all else fails to control symptoms conversion to a Billroth I anastomosis should be considered or a colonic implant plus vagotomy tried

The serious complication of gastro-jejuno-colic fistula (p 227) should be remembered as a cause of diarrhoea developing for the first time some months or years after gastro-enterostomy or gastrectomy

By contrast a few patients become constipated after gastrectomy or vagotomy and radiography may show gross *dilatation of* the small intestine or segmentation of the barium Kymography may reveal reduced activity or intestinal spasm and the spasms may be accompanied by *burning abdominal* pain The pathogenesis of these states is obscure and their treatment is unsatisfactory but sodium bicarbonate (4 g) with meals may be *helpful* (Glazebrook and Wrigley 1952)

Frank clinical steatorrhoea with frothy pale bulky and offensive stools is rare although after gastrectomy many patients pass motions which are paler than normal and most have an excess of fat in their faeces It may accompany severe intermittent bilious vomiting and may follow the rare accidents of injury to the pancreatic duct or anastomosis of the stomach to the ileum at operation Occasionally it follows the ingestion of ripe cheese which suggests a bacterial origin Steatorrhoea associated with lesions of the small or large intestine such as idiopathic steatorrhoea diverticula or ulcerative colitis may be unmasked or aggravated by gastric resection (Paulley *et al* 1957) Treatment is difficult Any recognisable cause should be removed if possible otherwise it should be treated in the same way as diarrhoea Conversion to a Billroth I anastomosis has given excellent results (Naish and Capper 1953)

DISTURBANCES OF NUTRITION

Loss of weight

Loss of weight is a common complaint after operations on the stomach It causes no serious disability (with the possible rare exception of pulmonary tuberculosis p 215) but is a matter of concern to the patient and is very difficult to remedy

It is important to realise that much weight is often lost *before* operation and that a complaint of loss of weight often means no more than that the patient has failed to regain his pre illness or health weight afterwards After sub-total Polya resection the majority of patients are seven to fifteen lbs below their health weights (Johnston *et al* 1958) but the overall effect of the operation is to cause an average *increase* of about three lbs (Wells and Welbourn 1951) The proportion of patients (about three quarters) who are below their standard weights (calculated from sex height and age) is about the same before operation as it is after (Baron 1954)

AETIOLOGY —Several factors influence the direction and extent of the change in weight after operation

Time after operation —The maximum fall occurs within the first few weeks of operation and is usually greater than that which follows other forms of major surgery It tends to become stabilised within twelve months although it continues to increase slowly for much longer in some patients (Fig 38)

Pre operative weight change (Johnston *et al* 1958) Nearly half the patients who come to surgery remain within fifteen lbs either way of their health weight up to the time of operation Afterwards they tend to *lose* a few pounds (Fig 38A) Most of the remainder lose a great deal of weight before operation and *regain* much of it afterwards (Fig 38B) Their final

weight however is below that of the previous group The few *patients who* gain much weight beforehand usually lose some of it afterwards but remain heavy (Fig 38c)

Sex—Women tend to lose weight more readily than men before and after operation (Fig 38) (Blake and Rechnitzer 1953 Anderson *et al* 1955)

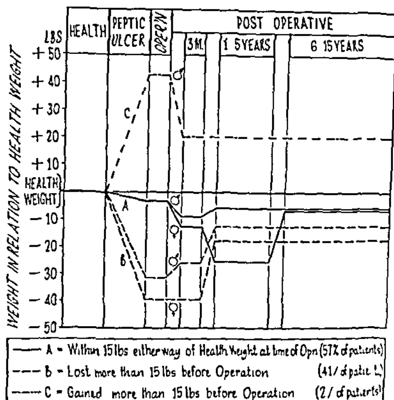


FIG 38
Effects of sub total Polya gastrectomy on body weight in relation to pre operative weight change sex and time after operation (Welbourn 1953)

Extent of resection—Loss of weight is unusual after gastro-enterostomy (Blake and Rechnitzer 1953) but is common after all types of gastrectomy the loss being directly proportional to the extent of the resection (*Pulvertaft* 1952 Welbourn 1953 Everson *et al* 1957b) After total gastrectomy marked loss of weight is the rule (Welbourn 1953) The addition of vagotomy does not usually influence the weight change so that a limited resection plus vagotomy causes less loss of weight than a high resection alone (Orr 1951)

Type of anastomosis—A Billroth I gastrectomy causes less loss of weight than a Polya (Meurling 1953 Moore *et al* 1953 Moroney 1953) A limited Billroth I resection combined with vagotomy causes less loss than vagotomy and gastro-enterostomy (Zollinger and Ellison 1954)

PATHOGENESIS —There are several contributory causes

Reduced calorie intake —Loss of weight is most marked in those whose intake of food is reduced by a poor appetite or by severe post-cibal symptoms Patients may be unaware that their appetites are poor until treatment improves them (see later) Dietary analyses show that those who have no post-cibal symptoms and whose weight is high eat on the average just about the amount that they require theoretically while those who have severe symptoms and whose weight is low only eat about three-quarters of their requirements This is the most important single cause (Johnston *et al* 1958)

Impairment of digestion and absorption is not an important cause in most cases because the loss of calories in the faeces is very small In patients who suffer severe steatorrhoea however it may be a significant factor If the capacity to eat is unimpaired the faecal loss can easily be compensated by the consumption of more food After total gastrectomy when the capacity is small and steatorrhoea is great it is not surprising that loss of weight is usually serious (Welbourn *et al* 1953)

Vomiting of food is not common but is sometimes a contributory factor

Loss of specific factors —Sometimes no obvious cause for loss of weight can be found and it may be that some metabolic factor is involved The basal metabolic rate has been studied only in the rat and in this species it is not increased by gastrectomy (Johnston 1958) Treatment of megaloblastic anaemia after total gastrectomy with vitamin B sometimes results in a marked increase in weight (Welbourn *et al* 1956)

TREATMENT —It is easier to maintain weight than it is to regain it after it has been lost and more attention should be paid to methods of increasing the intake of calories and of protein during the early post-operative period (Baron 1954 Lee 1957) The help of a skilled dietician should be enlisted with each patient until his weight is stabilised at its optimal level

Once loss of weight is established other serious causes such as tuberculosis or cancer must be excluded Reassurance that no harm will result should be given Modest gains in weight averaging about four lbs in eight weeks can be achieved by dietetic management or by the administration of methyl testosterone (25 mg per day sublingually) or iproniazid (Marsilid Roche —50 mg per day by mouth) (Johnston *et al* 1958) Rather better results (six lbs in eight weeks) follow the combination of methyl testosterone (50 mg per day) and diet Androgens should not of course be given to women but in these doses they are usually free from side effects in men The dietetic management is best provided by a dietician The aim is to increase the calorie and protein content of meals but not to increase their bulk Some patients require financial assistance Methyl testosterone produces an increased sense

of well being and often improves the appetite It encourages the anabolism of protein which should be provided in adequate amounts in the diet Iproniazid *increases the appetite* Operative treatment of severe symptoms by conversion to a Billroth I anastomosis (or by other methods) often causes a striking increase in weight (Capper and Welbourn 1955)

Iron deficiency anaemia

Iron deficiency anaemia develops very commonly after gastrectomy It frequently goes unrecognised and consequently causes much minor ill health Regular examination of the blood should disclose the anaemia before it becomes serious fortunately it is easy to correct

CLINICAL FEATURES —The anaemia is often insidious in onset and slowly progressive Lack of energy and dyspnoea on exertion are the commonest symptoms The former is noticed constantly or on exertion or towards the end of the day and not after meals as in the dumping syndrome Many patients have no subjective complaints but some declare themselves much improved after treatment Pallor is usual but the patient s colour may be deceptively good Koilonychia is not uncommon Angular stomatitis superficial glossitis and dysphagia (Paterson Kelly or Plummer Vinson Syndrome) are occasionally found in women and very rarely in men The mouth lesions however are more often caused by simultaneous deficiency of riboflavine (Welbourn 1953)

HAEMATOLOGY —The peripheral blood has the typical characteristics of an iron deficiency anaemia The mean corpuscular haemoglobin concentration and the serum iron level are low The leucocytes are normal The marrow shows normoblastic hyperplasia with inhibition of maturation but no megaloblastosis (Lyngar 1950 Baird *et al* 1959)

AETIOLOGY —Anaemia is rare in men before operation unless there has been haematemesis or melaena It is not uncommon in women (Fig 39) After operation several factors affect its incidence

Sex has a marked effect (Welbourn 1953 Blake and Rechnitzer 1953 Mercer 1954 Wallensten 1954 Baird *et al* 1959) (Fig 39) Women become anaemic more often and more severely than men Women under the age of fifty (*i e* those who are menstruating) are affected most of all and pregnancy following gastrectomy always causes severe anaemia

The *time after operation* affects the haemoglobin concentration (Fig 39) There may be temporary improvements but on the whole the level falls steadily as the years go by (Baird *et al* 1959)

The *type of operation* has some effect Anaemia is uncommon after gastro-enterostomy (Blake and Rechnitzer 1953) but very common after all forms of gastrectomy A Polya resection causes more anaemia than a

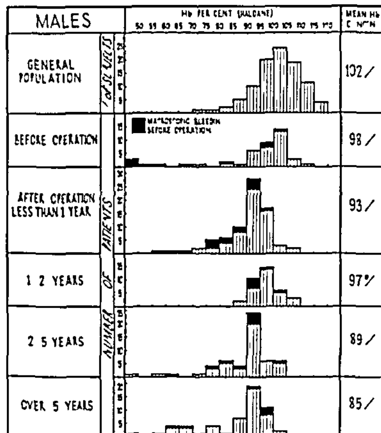


Fig 39A

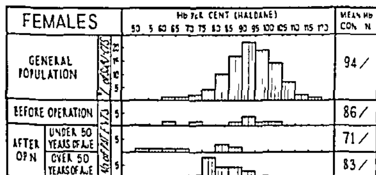


Fig 39B

Distribution of haemoglobin concentrations before and after sub total Polya gastrectomy (Welbourn 1953) The distribution for the general population is taken from Haemoglobin levels in Great Britain in 1943 (M R C 1945) A Males B Females (Larger series show that the haemoglobin levels fall progressively in women as well as in men)

Billroth I (Morley and Roberts 1928 Morley and Bentley 1938 Wallensten 1954 Baird *et al* 1959) but the extent of the resection is probably unimportant After total gastrectomy iron deficiency anaemia usually precedes the megaloblastic change Vagotomy and gastro enterostomy probably causes less anaemia than Polya gastrectomy (Smart and Williams 1958 Burge and Pick 1958)

The *site of the initial lesion* probably has a slight effect those with gastric ulcers being slightly more prone to anaemia than those with duodenal lesions (Watson 1947 Welbourn 1953)

Acute bleeding pre-operatively is a potent cause of anaemia immediately after operation (Morley and Roberts 1928) but there is usually a steady spontaneous improvement during the next year or two (Fig 39)

PATHOGENESIS —There are probably several factors which combine to cause anaemia after gastric operations The fact that it can usually be corrected by iron therapy indicates that a deficiency of iron is its immediate cause The body s stores of iron may be depleted by previous bleeding from peptic ulceration A deficiency of iron in the diet does not account for the anaemia in men and older women but may do so in women of child bearing years (Welbourn 1953 Baird *et al* 1959) The absorption of organic iron from the diet is impaired after gastrectomy (p 181) Bleeding from the uterus or elsewhere aggravates the tendency to anaemia and the body s increased need for iron during pregnancy and lactation has the same effect There is no evidence that occult bleeding from recurrent ulcers is a major cause of anaemia (Hartfall 1934 Larsen 1934 Watson 1947 Welbourn 1953 Baird *et al* 1959) Protein deficiency is a possible aggravating factor in the rare cases which do not respond completely to iron therapy by any route (Rekers *et al* 1943)

PREVENTION AND TREATMENT —Active measures must be taken to maintain a normal haemoglobin concentration at all periods before during *and after* operation and blood transfusion should be used if necessary

The diet should include adequate amounts of iron-containing foods such as liver kidney meat eggs and lentils It has been suggested that iron should be given prophylactically at regular intervals after operation (Farris *et al* 1943 Capper 1951) but there is no exact information about its effectiveness The blood must certainly be examined routinely during the follow up of patients and the interval between examinations should not exceed one year Particular care should be taken with pregnant *women* All pathological bleeding from gums piles etc and excessive menstrual loss must receive attention

If anaemia develops iron should be given by mouth Ferrous gluconate (300 to 600 mg t d s) ferrous succinate (150 mg to 300 mg t d s) and sodium ironedetate (Sytron Parke Davis—380 mg t d s) are tolerated better than the older preparations and are therefore more likely to be taken by the patient Ascorbic acid (50 mg) aids the absorption of iron and the response may be more rapid if it is given with each dose (Moore and Dubach 1956) Anaemia is rapidly corrected in most cases and clinical improvement is striking Treatment should be continued for six to eight weeks after a normal haemoglobin level has been achieved Some patients relapse rapidly when treatment is stopped and must be maintained indefinitely on small doses (Welbourn 1953)

A few patients especially women fail to respond to iron by mouth or are intolerant of it Iron given intramuscularly usually causes dramatic improvement (Welbourn 1953 Baird *et al* 1959) and it should be given without hesitation in correctly calculated doses It may be possible to provide a store of iron which will prevent anaemia for some time

Very rarely there is an incomplete response to iron given by any route The reason is not clear but a high protein diet is worthy of trial

Megaloblastic anaemia

AETIOLOGY —Macrocytic anaemia clinically and haematologically in distinguishable from pernicious anaemia has been reported many times following operations on the stomach After gastro-enterostomy partial and sub total gastrectomy the incidence is very low indeed and is probably no higher than it is in the general population In many reported cases the anaemia can be attributed to other lesions in the alimentary tract or to a dietary deficiency (Welbourn *et al* 1956) It has been claimed (MacLean 1957) that atrophy of the gastric mucosa is found pre-operatively in those who subsequently develop megaloblastic anaemia After total gastrectomy megaloblastic anaemia probably develops in every patient who lives long enough and who receives no prophylactic therapy (Bethell *et al* 1945) It follows operation for benign as well as malignant conditions It is found in at least one third of patients within three years (MacDonald *et al* 1947) and the incidence increases steadily thereafter (Fellinger 1950) The small fringe of stomach which is sometimes left at the cardiac end to facilitate the anastomosis does not prevent its development (Welbourn *et al* 1956) Vagotomy does not appear to cause megaloblastic anaemia

CLINICAL FEATURES —The clinical features are similar to those of iron deficiency anaemia except that superficial glossitis and angular stomatitis are commoner and sub acute combined degeneration of the cord may develop

HAEMATOLOGY —Haematologically the onset is often insidious and peripheral macrocytosis (M C V up to 120 cu μ) with a relatively high haemoglobin concentration (*e g* 12 0 g /100 ml) may develop and persist for many months before the bone marrow becomes frankly megaloblastic Intermediate megaloblasts however are usually present from an early stage (Welbourn *et al* 1956) Occasionally the onset is sudden Leucopenia and a high serum bilirubin are not uncommon

PATHOGENESIS —The most likely cause of the megaloblastic anaemia is the removal of the whole stomach which is the only source of intrinsic factor in man (p 182) In some cases there may also be a deficiency of folic acid associated with abnormal alimentary function (Welbourn *et al* 1956)

A rare cause of megaloblastic anaemia after partial gastrectomy or gastro enterostomy is the presence of an intestinal cul de sac in which bacteria abound (Naish and Capper 1953) In these cases vitamin B_{12} is not absorbed even when it is given with intrinsic factor (Adams 1958)

PREVENTION AND TREATMENT —Since megaloblastic anaemia is almost certain to develop after total gastrectomy prophylactic therapy in the form of an injection of 50 μg of vitamin B_{12} should be given every two weeks After partial gastrectomy the routine use of liver preparations vitamin B_1 or folic acid is *not* required

If megaloblastic anaemia develops it must be investigated and treated with full haematological control Vitamin B_1 given parenterally in a dosage of 100 μg on alternate days for one week weekly for one month and every two weeks *thereafter* causes striking clinical improvement and restores the blood to normal within three months Occasionally some degree of peripheral macrocytosis persists for rather longer If a response is not obtained or is sub-optimal folic acid by mouth iron and ascorbic acid should be given in turn in addition to vitamin B_1 All have been effective on occasion Folic acid must never be given without vitamin B_{12} or liver extract because it affords no protection to the spinal cord An adequate intake of food must be assured

The rare cases which are associated with a cul de sac can be improved with antibiotics but should be treated surgically (Naish and Capper 1953 Adams 1958)

Vitamin B-complex deficiency states

These conditions are much commoner than is generally recognised As a rule they cause minor symptoms only *and are often overlooked* (Welbourn *et al* 1951) They may often be detected biochemically in the absence of clinical manifestations (Turnock and Welbourn 1953) Their incidence has

not been compared with that in the general population but it is much higher after operation than it is before They are recognisable in about thirty three per cent of women and five per cent of men at some time after a sub-total Polya resection (Welbourn 1953) and sometimes after other gastric operations Their incidence appears to be related to the extent of the resection (Blake and Rechnitzer 1953) The syndromes sometimes occur alone and sometimes in combination

Riboflavine deficiency is the commonest syndrome It causes angular fissures of the lips which may be unilateral or bilateral and superficial glossitis (Fig 40) The tongue is affected first at the tip and sides and sometimes all over the dorsum It is sore and smooth and the fungiform papillae may be prominent It is usually red but may occasionally be magenta-coloured or pale The lip and tongue lesions may develop independently or together The onset is sometimes acute but more often chronic the lesions being present continuously or intermittently for years Similar mouth lesions occasionally develop in iron deficiency or megaloblastic anaemia or are caused by dentures and these causes should always be excluded Occasionally there is no obvious cause for the lesions and treatment is of no avail



FIG 40
Angular stomatitis caused by riboflavine deficiency following gastrectomy The lesions are usually as shown here but may be more severe

Thiamine deficiency develops in several forms The commonest is incipient peripheral neuritis in which pins and needles muscle cramps or excessive sensitiveness are complained of in the limbs It is sometimes associated with a general feeling of fatigue similar to that caused by anaemia There are no abnormal neurological signs Fatigue alone is not uncommon after gastrectomy but it rarely responds to thiamine and its cause is not known In rare cases frank peripheral neuritis follows with or without oedema Very rarely Wernicke s encephalopathy and other disturbances of consciousness may develop

Pellagra is a rare sequel to gastric operations and requires no special description

PATHOGENESIS —The majority of patients who develop riboflavine deficiency and incipient peripheral neuritis are taking normally adequate amounts of B vitamins Some of them however and all who develop severe

deficiency syndromes are eating either too little food or an unbalanced diet. It is uncertain what other mechanisms are responsible.

PREVENTION AND TREATMENT.—Patients should be encouraged to eat wholemeal bread and other vitamin-containing foods. Riboflavine deficiency and incipient peripheral neuritis usually respond well to specific oral therapy. It is wise to treat them both with B-complex preparations since there may be sub-clinical deficiency of more than one vitamin and the administration of one alone may precipitate deficiency of another (Frazer 1949). At least 5 mg of both thiamine and riboflavine should be given daily in divided doses. The response is usually good and follows within days or weeks. It may be necessary to give supplementary vitamins indefinitely. Established neuritis cannot be cured, but vitamins should be given to stop it progressing. Encephalopathy and pellagra can be cured or much improved by intensive treatment with large doses of vitamins parenterally.

Severe malnutrition

The serum protein concentration is rarely affected much by partial gastrectomy (Welbourn 1953, Mercer 1954). There is a tendency, however, for those who have lost much weight to have low normal levels. Total gastrectomy is more often followed by hypoproteinaemia (Pack *et al.* 1947) and may cause reversal of the albumin globulin ratio (Tomodo 1952).

Oedema is uncommon after gastrectomy and when it occurs it is not always possible to find its cause. Recognisable causes are mild thiamine deficiency, severe anaemia and minor degrees of ' hunger oedema ' (see below). Cardiac, renal and local causes (including cannulation of ankle veins at the time of operation) must be excluded and specific treatment given.

Very occasionally the minor degrees of undernutrition and malnutrition which have been described progress to a much more serious state of affairs akin to that found under conditions of famine. The patient exhibits weakness, emaciation, gross oedema, anaemia and signs of vitamin deficiencies, and often suffers from diarrhoea and steatorrhoea. The serum proteins and lipids are low and the nitrogen balance is negative (Lambling and Conte 1949, Lambling *et al.* 1949, Naish and Capper 1953). Dietary deficiencies and alimentary dysfunction probably contribute to the development of the syndrome.

The condition is difficult to treat, and other serious lesions, such as gastro-jejuno-colic fistula, gastro-ileostomy and carcinoma should be excluded. A high calorie, high protein diet, vitamins, iron and blood transfusion often cause improvement. Some excellent results have been achieved by conversion of a Polya to a Billroth I anastomosis (Moroney 1953, Naish and Capper 1953).

PULMONARY TUBERCULOSIS CARCINOMA OF THE STOMACH AND CORONARY THROMBOSIS

Pulmonary tuberculosis

Pulmonary tuberculosis has been reported in two to four per cent of patients who have undergone gastrectomy and may account for the deaths of nearly a third of those who die some years after operation

It is found particularly in those who suffer severe post-cibal symptoms and who are under nourished (Pulvertaft 1952 Pearson 1954 Anderson *et al* 1955) The operation itself however is not necessarily a predisposing cause of the disease and it is likely that the pre-operative state of the patient plays a large part in determining its development Thus in one careful study (Thorn *et al* 1956) at least a third of the men and all the women who apparently developed tuberculosis after gastrectomy had abnormal chest X rays before operation and the disease was far commoner in those who were seriously underweight *before* operation than in those whose weights were normal Moreover the incidence was higher in those with ulcers in the stomach (especially the upper third) than in those with lesions in the duodenum

It is important to exclude active pulmonary tuberculosis in every patient for whom surgery is considered and if it is present to treat it before operation Very rarely (in pyloric stenosis etc) an operation may be necessary to ensure adequate nutrition in a patient with pulmonary tuberculosis In such cases the Polya operation should be avoided because of its liability to disturb alimentary function After operation chest X rays should be taken regularly in patients who are much underweight

Carcinoma of the stomach

Carcinoma of the stomach has been reported many times in patients who have undergone gastro-enterostomy or partial gastrectomy (Freedman and Berne 1954) Recent reports indicate that it develops more commonly in those whose stomachs have been partially resected than in the general population One large series showed that in those who had had duodenal ulcers the incidence was probably *no* higher but that in those who had had gastric ulcers it was about three times greater than expected in both sexes (Helsingen and Hillestad 1956) In another report (in which no distinction was made between gastric and duodenal ulcers) the mortality rate from gastric carcinoma after gastrectomy was found to be double that in the general population (Krause 1957) The cause of the increased incidence is not known nor is it clear what relation if any malignancy may bear to the other pathological changes

which may be recognised after operation by gastroscopy and gastric biopsy (p 184) The average interval between operation and the recognition of the disease is about twenty years (Freedman and Berne 1954 Helsingen and Hillestad 1956) The clinical features are those of any gastric carcinoma When the lesion develops at or near the stoma it must be distinguished from peptic ulceration and if there is any doubt about the innocent nature of an ulcer a laparotomy must be performed without delay The stomach should be resected if possible

Coronary thrombosis

Recently evidence has been presented that partial gastrectomy for duodenal ulcer lessens the risk of the patient subsequently developing coronary thrombosis (Walker *et al* 1958) In a very long term follow up study (Krause 1957) it was found that one third of the late deaths following gastrectomy were the result of cardio vascular disease Patients with untreated peptic ulcers have a higher incidence of coronary artery disease than have normal subjects *(Watkinson 1956) The exact relationship between gastric and duodenal* ulceration and their surgical treatment on the one hand and coronary atheroma and thrombosis on the other must await the results of further follow up studies in which the age of the patient the site of ulceration and type of operation are clearly defined

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CHAPTER XIII

RECURRENT ULCERATION

By Richard B Welbourn

THE term recurrent ulceration is used to include all forms of peptic ulceration which persist or recur after operation either at the original site or elsewhere Recurrence after an operation for *gastric* ulcer is nearly always situated in the stomach When it follows operation for *duodenal* ulcer its site depends on the nature of the operative procedure After gastro-enterostomy a duodenal ulcer nearly always heals unless the stoma becomes obstructed (Tanner 1954) any recurrence develops at the gastro-jejunal stoma (gastro-jejunal stomal or marginal ulcer) or within the first two inches of the efferent jejunal loop (jejunal ulcer) Very rarely it develops in the afferent loop After a Polya gastrectomy a duodenal ulcer always heals even when the antrum has not been resected (Wells and Brewer 1948) but a recurrent ulcer may develop at the same sites as those following gastro-enterostomy After a Billroth I resection the recurrence is in the first part of the duodenum at the stoma or on the lesser curvature of the stomach adjacent to the stoma (Goligher *et al* 1956)

Incidence

Recurrent ulceration was met frequently after the older operations particularly after gastro-enterostomy for duodenal ulcer but is now happily uncommon It is difficult for several reasons to establish the precise incidence Some published reports have been made too soon after operation to be reliable some do not give an adequate analysis of the factors which influence the recurrence rate and others do not make any allowance for the possible development of recurrent ulcers in patients who have not been traced Many authors fail to state their criteria for the diagnosis of recurrence some have listed only the proved cases while others have included all those that were suspected clinically A classification into three categories— possible probable and proved —will be suggested later after discussion of the clinical features and diagnosis (p 223) These shortcomings must be borne in mind in the analysis which follows The incidence depends on several factors among which is the site of the original ulcer

Gastric ulcer —Recurrence is much less common after operations for gastric ulcer than after those for duodenal lesions if the main ulcer bearing portion of the stomach (lesser curvature and pyloric antrum) is resected It is

perhaps more frequent after Billroth I than after a Polya operation (Comfort *et al* 1957) The reported incidence ranges from 0 1 per cent (one out of 1 000 Tanner 1954) to 7 5 per cent (three out of forty Douglas 1947) The average for all published series is about one per cent Gastro-enterostomy was abandoned long ago in the treatment of gastric ulcer because the lesion often persisted after operation

DUODENAL ULCER —The recurrence rate following operations for duodenal ulcer is influenced by the nature of the operation by the sex of the patient and by the length of time which has elapsed since operation

Nature of the operation

The nature of the operation can be considered under four headings namely the extent of resection the type of anastomosis the presence or absence of the pyloric antrum and vagotomy

Extent of resection —The recurrence rate varies inversely with the extent of the resection The following average figures were obtained from the pooled results of several published series (Ivy *et al* 1951) All the operations involved a gastro-jejunal anastomosis

Amount of stomach resected	*Recurrence rate*
0 (gastro enterostomy)	14 5%
55 70%	5 8%
75%	3 5%

The true rate for gastro-enterostomy is almost certainly higher than this and recent reports have placed it around fifty per cent (Clark 1951 Tanner 1954) No reliable method has been found for recognising before operation the patients who are likely to develop recurrent ulcers Measurement of gastric secretion is of little help (Kay 1955) and the appearance of the gastric mucosa at gastroscopy has proved disappointing (Taylor 1954)

The rate following three-quarters resection may well be as low as two per cent (Tanner 1954 Capper and Welbourn 1955 Goligher *et al* 1956) Very high resections reduce the rate almost to zero (Visick 1948 Pulvertaft 1952) although there are rare cases of stubborn persistent ulceration which yield only to total gastrectomy (Balint *et al* 1957 Balint and Gummer 1958)

Type of anastomosis —A *Billroth I* gastrectomy is much less effective than a Polya in controlling ulceration (Wallensten 1954 Capper and Welbourn 1955) Recurrence rates in the region of fifteen to thirty per cent have been reported after three-quarters or higher resections (Ordahl *et al*

1955 Golicher *et al* 1956) Operations of the Billroth I type modified by the insertion of a length of *jejunum* (Henley 1953) between the stomach and the duodenum are very liable to cause jejunal ulceration (Jones 1954 and Maingot 1954 personal communications) A length of transverse *colon* used similarly (Moroney 1953) also develops ulceration but probably less commonly The *Roux-en-Y* anastomosis combined with gastro-enterostomy was abandoned long ago because of the frequency with which stomal ulceration developed (Ivy *et al* 1951) It has recently been re introduced in association with gastrectomy *without* vagotomy Preliminary data indicate that the recurrence rate is at least eight per cent (Pulvertaft 1958 personal communication)

Role of pyloric antrum—Failure to remove the mucosa of the pyloric antrum (which secretes gastrin) causes stomal ulceration to develop in nearly forty per cent of Polya resections when up to three-quarters of the body of the stomach are resected (Ivy *et al* 1951) Very high resections appear to reduce the risk of recurrence (Visick 1948) Preliminary reports indicate that the recurrence rate following radical segmental resection (in which the antrum is anastomosed to the fundus) carries a very low recurrence rate (MacLean *et al* 1953)

Vagotomy—When vagotomy *alone* was performed for duodenal lesions the ulcer persisted or recurred in about ten per cent of cases (Ivy *et al* 1951) and ulcers developed in the *stomach* in a few cases (Johnson and Orr 1954) The operation is now always combined with some procedure which permits the stomach to empty The reported incidence of recurrent or stomal ulceration following vagotomy and gastro-enterostomy varies from one per cent of proved cases (Pollock 1952 Holt and Robinson 1955) to over twenty five per cent of clinically suspected lesions (Jordan *et al* 1952) The true figure may be around ten per cent (Bennett Jones and O Domhnaill 1955 Henson and Rob 1955) and is almost certainly higher than that following a three quarters Polya resection Vagotomy combined with limited (half to two-thirds) partial gastrectomy either of the Polya or Billroth I type appears to control ulceration as effectively as a high Polya gastrectomy alone but the evidence is incomplete (Fallis and Barron 1949 Johnson and Orr 1954 Moloney 1954 Bennett Jones and O Domhnaill 1955 Coffey and Lazaro 1955 Edwards *et al* 1957) The effectiveness of vagotomy depends to a large extent on its completeness (Dragstedt *et al* 1947) It may well be greater when a simple and reliable method has been found for assessing its completeness at the time of operation (Burge and Pick 1958)

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Sex

Recurrent ulceration is a greater problem in men than it is in women After gastro-enterostomy severe recurrence is much more frequent in men although the total relapse rate is about the same in the two sexes (Clark 1951) After gastrectomy recurrence is at least three times commoner in men than in women after either a Polya or a Billroth I resection (Wallensten 1954)

Time after operation

The majority of recurrent ulcers develop within the first few years after operation After gastro enterostomy from one third (Clark 1951) to two-thirds (Ivy *et al* 1951) develop within two years After gastrectomy two-thirds appear within five to six years (Wallensten 1954) It is important to realise however that recurrence may develop as late as twenty years after operation and this is especially true of recurrence following gastro-enterostomy (Davey 1959)

Age at operation

After gastro enterostomy the recurrence rate is higher in those under forty years of age at the time of operation than in older patients (Clark 1951) In the latter however it is still prohibitively high whether done electively or for the relief of pyloric stenosis

Clinical features and diagnosis

The commonest symptom of recurrent ulcer is pain similar or identical to that which was experienced before operation It is often felt at the site of the ulcer so that in the case of gastro-jejunal ulcer following Polya gastrectomy it may be in the left hypochondrium The pain must be distinguished by careful questioning from that of other conditions such as severe post-cibal fullness intestinal colic and hiatus hernia There are usually exacerbations and remissions similar to those of primary peptic ulcer Nausea and vomiting diarrhoea and loss of weight are occasional symptoms Haematemesis melaena and perforation occur as frequently with recurrent ulcers as they do with primary ulcers and they may appear without any previous pain Stenosis of the stoma by an ulcer (analogous to pyloric stenosis) is rare Gastro-jejuno-colic fistula is a serious but uncommon complication which will be discussed later The physical signs are often minimal Tenderness is inconstant and is sometimes present when there is no ulcer Very rarely an inflammatory mass may be palpable The relative frequency of symptoms and complications in one series of 200 cases was as follows (Marshall and Terrell 1957)

Symptoms	Pain	80
	Nausea and vomiting	35
	Diarrhoea	17
	Loss of weight	38
Complications	Bleeding	48
	Perforation	10.5
	Obstruction	6
	Gastro-jejuno-colic fistula	17

Special investigations are less helpful than they are in patients with intact stomachs. A barium meal is often unsatisfactory because the stomach empties rapidly and cannot be adequately outlined, and because post-operative scarring produces irregularities which may be mistaken for ulcer craters. False negative results are common (Walters *et al.* 1955; Marshall and Terrell 1957). Considerable help can be obtained from the injection of morphia (5 mg. intravenously) immediately before the meal (Crone 1954). This causes the stoma to contract and allows the stomach to be distended by the barium, so facilitating the examination. Gastroscopy may reveal an ulcer in the stomach or on the gastric edge of the stoma. Hyperaemia at the stoma may suggest the presence of an ulcer just beyond the field of vision. An ulcer in the duodenum or in the jejunum cannot be excluded by gastroscopy. The presence of free acid in the gastric juice, especially after high Polya resection, is suggestive of ulceration. Blood in the gastric juice nearly always indicates ulceration. After vagotomy an insulin test meal (Hollander 1946) provides information about the completeness of the operation, and ulceration is more likely to be present if free acid is found than if it is not. The results of the investigations may be equivocal and surgical exploration may be required before a final diagnosis can be made. It should be borne in mind that an ulcer in the stomach may be malignant.

It is convenient to classify recurrent ulcers under three headings:

Possible—a suggestive history, free acid in the gastric juice.

Probable—probable ulcer crater on X-ray, hyperaemia of the stoma on gastroscopy, blood in the gastric juice or frank haemorrhage.

Proved—definite crater on X-ray, seen at gastroscopy, found at laparotomy, perforated.

It would be helpful if some such criteria were specified when the results of operations were reported.

Treatment

Treatment of recurrent ulcer by medical means is usually disappointing (Balint *et al.* 1957). It is worthy of trial in patients whose symptoms are

mild or in whom they develop only on rare occasions Otherwise all those with probable or proved ulcers and those with possible ulcers whose symptoms are severe should be subjected to further operation Those who develop recurrence after one operation are much more liable to do so again after a second than are patients with primary ulcers who undergo the same (second) operation (Wells 1954 Balint *et al* 1957) The difficulties and dangers increase with every operation and gastric re resection may carry a mortality rate as high as fifteen per cent (Walters *et al* 1955) For this reason every effort must be made to procure permanent freedom from ulceration when an operation is performed for recurrence When the ulcer is in the stomach this involves a more radical (three quarters) resection with a Polya anastomosis Total gastrectomy may extremely rarely be required Care must be taken to ensure that the ulcer is not malignant (Gray and Lofgren 1949) When the recurrence is at the stoma in the duodenum or in the jejunum there are four requirements

1 Complete removal of the pyloric antrum
2 Complete vagotomy
3 Resection of three quarters of the body of the stomach and
4 A Polya anastomosis

Any of these procedures which have not been done already must be undertaken (Fig 41) These rules should be broken only if severe post-cibal symptoms make it desirable to employ a Billroth I or a Roux-en Y anastomosis The operation may be performed electively or as an emergency at the time of perforation or bleeding If in an emergency the patient is not fit for a major procedure the minimum that will save life should be done and an elective operation performed as soon as possible The vagotomy can usually be done *via* the abdomen together with whatever other surgical procedures are necessary It should be done through the chest if a previous abdominal vagotomy has been shown to be incomplete or if severe adhesions between the stomach and the diaphragm render the abdominal operation unduly hazardous It is not possible of course to verify the presence of an ulcer nor to exclude malignancy when the thoracic route is used Some surgeons however when dealing with a recurrent ulcer after a Polya gastrectomy as a first step simply perform a transthoracic vagotomy which is often successful only later if symptoms persist do they carry out an abdominal exploration and higher gastric resection (Wells 1959 personal communication)

There remains the rare problem of the patient who develops a recurrent ulcer when all four requirements have been met The first essential is to exclude an islet-cell tumour of the pancreas especially one of the non insulin

secreting type (Zollinger and Ellison 1955 Zollinger and McPherson 1958) This is usually associated with gross gastric hypersecretion and the ulcers tend to recur rapidly after normally adequate operations The only sure method of treatment is total gastrectomy (with a Roux-en Y anastomosis) which should be combined if possible with excision of the tumour Total gastrectomy may very rarely be required for the treatment of stubborn ulceration in which no such association can be found (Balint and Gummer 1958)

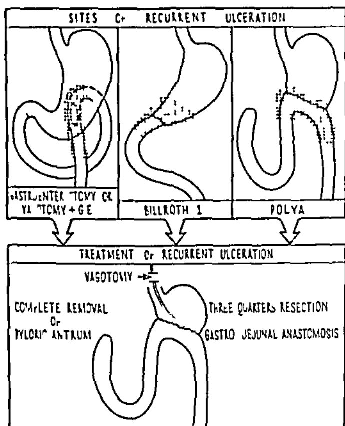


FIG 41
Sites at which ulcers may recur after operations for duodenal ulcer (hatched areas) and operation for treatment of recurrent ulceration

OPERATION —The first essential is to find out from operation notes X rays etc as much as possible about the previous operations Multiple adhesions may render the dissection tedious and it is helpful for the surgeon to know what abnormal anatomy to expect If there has been only one previous operation it is usually convenient to enter the abdomen through the old incision If there have been several an inverted V shaped or double Kocher incision (Wells and Brewer 1948) facilitates the entry into the peritoneal cavity and gives excellent access

After posterior gastro-enterostomy the first step is to find the afferent and efferent jejunal loops and to trace them to the stoma The dissection is started below the transverse mesocolon and continued up as far as possible Great care must be taken to avoid damage to the middle colic vessels which may lie close to the stoma and be involved in an inflammatory reaction around the ulcer The next step is to enter the lesser sac by dividing some of the vessels between the stomach and the gastro epiploic arch Some adhesions behind the stomach usually require division before the stoma is reached and the stoma may be adherent to the transverse colon When the stoma and the jejunal loops have been exposed and mobilised they are examined and palpated carefully An ulcer may be visible as a red patch or as an inflam matory mass It may feel thickened and an ulcer crater can sometimes be identified with the finger If there is doubt about an ulcer being present the anastomosis should be opened at the front and the stoma inspected from within Sometimes an ulcer is found to have healed temporarily but the scar is usually recognisable

If there is no sign of past or present ulceration and the clinical features are indefinite the stoma should be repaired and the abdomen closed Other wise vagotomy and gastrectomy should be undertaken The jejunum is severed from the stomach and care is taken to ensure that no gastric mucosa is left attached to it The hole in the jejunum is then closed transversely (to avoid narrowing of the lumen) or if this is not practicable the loop on which it lies is resected and continuity is re established by end to end anastomosis In either case the repaired region of the jejunum will form part of the afferent loop after gastrectomy The opening in the stomach is closed temporarily with a clamp or with stitches Vagotomy and gastrectomy are then performed as in a primary operation The hole in the transverse mesocolon is either closed or used for a new retro colic anastomosis

After Polya gastrectomy a similar procedure is used for exposing the stoma The operation is usually easier with an ante-colic than with a retro-colic anastomosis The duodenal stump must be identified mobilised and examined carefully If any antral wall is found it must be excised and the stump re-closed It is convenient to leave the jejunal loops attached to the stomach while the vagotomy is being done and while the gastric stump is being mobilised for higher resection The jejunal opening may be used for the new stoma or dealt with in the manner described already

For recurrent ulceration following a Billroth I gastrectomy a Polya operation is required and is so similar to a primary gastrectomy of that type that no special description is necessary

GASTROJEJUNOCOLIC FISTULA

This dire complication of stomal or jejunal ulcer is mostly fatal if it is not treated by bold surgery and active supportive measures The difficulties and dangers of operation are considerable and the result is not always satisfactory

After gastro-enterostomy or Polya gastrectomy the stoma and jejunal loops lie close to the transverse colon If an ulcer forms at the point of contact it may erode the wall of the colon and form a fistulous communication between the colon and the stomach or jejunum or between all three The commonest site for the fistula is between the colon and the efferent jejunal loop (Lowdon 1953) The result is that the highly infected and irritating contents of the colon pass into the stomach and jejunum and cause inflammation and marked intestinal hurry (Bolton and Trotter 1930 Pfeiffer and Kent 1939 Renshaw *et al* 1946) The passage of food from the stomach into the colon occurs to a much smaller extent and is relatively unimportant

Incidence

The reported incidence of fistula formation in patients with stomal ulcers varies from nine per cent to twenty two per cent (Marshall and Knud Hansen 1957) These estimates are probably too high because they take no account of patients with stomal ulceration who do not return to hospital Most cases follow posterior gastro-enterostomy although some are found after anterior gastro-enterostomy or partial gastrectomy with an anterior or a posterior anastomosis (Lowdon 1953) Most fistulae develop two to fifteen years after operation but some appear within six months and others not until over twenty years afterwards

Clinical features and diagnosis

The condition is almost confined to men in whom at least ninety-eight per cent of cases are found the commonest age at which it develops is between thirty five and fifty five but no age group is immune (Lowdon 1953) Symptoms are variable often develop suddenly and are usually persistent although some patients enjoy periods of freedom The commonest symptom is *diarrhoea* which develops in over eighty per cent of cases The patient may pass from three or four to twenty or thirty motions in the day The faeces are usually soft or fluid contain mucus and are pale They sometimes contain recently ingested food *and steatorrhoea is common There may be associated* borborygmi and faecal incontinence A fistula should be suspected in any patient who develops persistent diarrhoea some time after operation *Loss of*

weight which may be rapid and severe is almost as common as diarrhoea and may cause emaciation *Vomiting of faecal material* or eructation of faecal smelling gas is pathognomonic of the condition but less frequent If the patient is given an enema he may vomit some of it Vomiting or eructations of a non faecal type sometimes occur Abdominal *pain* is present in about half the cases but is rarely of the ulcer type Indeed ulcer pain may disappear when the fistula forms Pain may develop in the lower part of the abdomen and take the form of a dull ache colic or a feeling of fullness after meals *Bleeding* from the site of the fistula is an occasional symptom *Nutritional and metabolic disorders* other than loss of weight may be serious Iron-deficiency anaemia is usual although haemoconcentration may increase the haemoglobin concentration Hypoproteinaemia is frequent and may cause oedema which is sometimes severe Hypocalcaemia is not uncommon and occasionally causes tetany Dehydration and deficiency of electrolytes are common and vitamin deficiencies of any type may develop

All these disturbances combine to cause very serious illness and most patients seek hospital treatment within six months of the onset of symptoms Some patients are so tired weak breathless and wretched that they become bed ridden A very few tolerate their fistulae for long periods without developing any of the characteristic features of the disease

Physical signs apart from those which have been mentioned already are few and half the patients have normal abdominal findings Tenderness distension increased peristalsis and an inflammatory mass may sometimes be found

The most reliable test for the demonstration of a fistula is a barium enema which shows the passage of contrast into the stomach or jejunum in nearly 100 per cent of cases A barium meal shows passage of contrast into the colon in only about forty per cent Gastroscopy and test meals are of little help Occasionally a fistula is found unexpectedly during an operation for stomal ulcer

Treatment

Surgical closure of the fistula and an operation to prevent recurrence of ulceration are required to cure the condition (Lowdon 1953 Marshall and Knud Hansen 1957) This is a formidable undertaking in an ill patient Modern pre operative measures have improved the outlook considerably but there remain a few patients who require staged procedures

Pre-operative treatment should include transfusion with whole blood and blood plasma replacement of water salt (including potassium) and calcium deficiencies and the correction of any acid base disturbance A low residue diet with a high calorie protein and carbohydrate content is given but oral

fluids should be restricted Large doses of all the vitamins are given parenterally If a polythene tube is passed into the vena cava a fifty per cent solution of glucose can be infused without danger of thrombophlebitis and supplies much needed calories The stomach should be washed out once daily and the bowel prepared with neomycin and sulphonamide These should be given in an emulsion and not in capsules or tablets which may be swept through the bowel undissolved Opium helps to reduce the bowel motility This treatment pursued vigorously for two to three days immediately before operation produces considerable improvement If it is prolonged the patient may deteriorate again because of the continued patency of the fistula

If the patient is too ill to withstand major surgery at once a palliative operation must be undertaken as a temporary measure to prevent the contents of the colon from entering the stomach and jejunum There are two alternatives both of which are effective (Fig 42) The simpler is to make a proximal defunctioning colostomy in the ascending (Pfeiffer and Kent 1939) or transverse (Lowdon 1953) colon The main disadvantage of this procedure is that a third operation is required for its closure The second alternative is to divide the terminal ileum close its distal end and make an anastomosis between the proximal end and the pelvic colon (Lahey and Marshall 1943) This is a bigger undertaking than a simple colostomy and has the disadvantage that the proximal half of the colon must be removed at the second operation Both these procedures carry a low mortality rate and rapidly abolish the symptoms and disturbances caused by the fistula They do not however cure the ulcer and a definitive operation must be undertaken as soon as the patient is fit enough for it The usual time will be two to three months In an old or otherwise unfit patient who remains free from ulcer symptoms it may be justifiable to postpone the second operation indefinitely (MacLeod and Galloway 1957)

A definitive operation may be undertaken at once in suitable cases or as the second operation in a staged procedure Technically it may be very difficult indeed and it should not be attempted by any but an experienced gastric surgeon The operation involves ideally complete vagotomy removal of all the pyloric antrum three quarters resection of the stomach and a Polya anastomosis The fistula must be divided and the hole in the colon closed Sometimes resection of the fistulous part of the colon is necessary continuity is re-established by end to-end anastomosis If ileo-colostomy was performed previously right hemi-colectomy is combined with the gastrectomy and the severed end of the colon is closed This increases the magnitude of the operation If a colostomy is present it is isolated carefully from the operation field and closed later

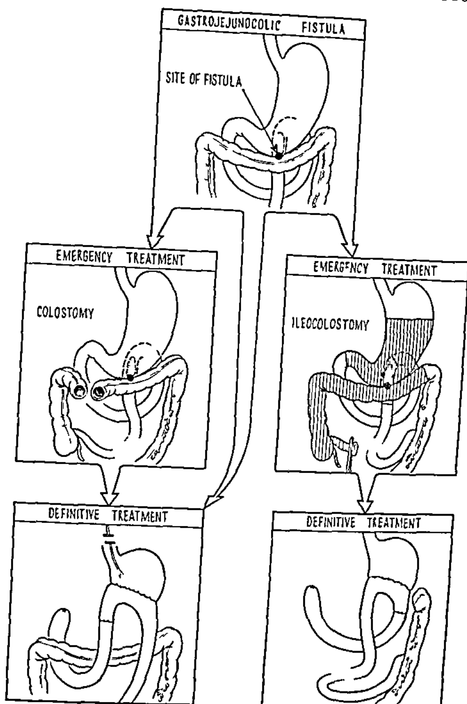


FIG 42
Treatment of gastrojejunocolic fistula Left standard method Right Lahey's procedure (The shaded area is resected at the definitive operation)

Up to about ten years ago the mortality rate of operations for fistula was extremely high and recurrent ulceration sometimes with recurrent fistula was common The present position is illustrated by two series of cases which have been published recently (Walters et al 1955 Marshall and Knud Hansen 1957) A one stage operation was performed in most patients but the high gastrectomy and vagotomy (advocated here) were performed in only a few The results were as follows

Total number of patients	38
Operative deaths	0
Patients followed	33
Satisfactory	26
Died later of disease	2
Recurrent fistula	2
Recurrent ulcer (not fistula)	3

More radical operations would probably have reduced the recurrence rate The solution to the problem however lies in its prevention by adequate primary surgery

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APPENDIX

ENGLISH TRANSLATIONS OF EARLY GERMAN CONTRIBUTIONS TO GASTRIC SURGERY

APPENDIX

Billroth Th (1881) Offenes Schreiben an Herrn Dr L Wittelshofer (Open letter to Dr L Wittelshöfer) *Wien med Wschr* 31, 162 165 (No 6)

Vienna 4th February 1881

Dear Colleague

I willingly accede to your request for information about the resection of the stomach which I carried out on January 29th this year since it concerns the very important question whether it is possible to cure that very common disease carcinoma of the stomach by surgical intervention

Seventy years have passed since a young physician Karl Theodor Merrem published a dissertation in which he demonstrated by means of experiments on does that the pylorus could be excised and the stomach joined to the duodenum and that two out of three dogs survived the operation He was bold enough to suggest that this operation should also be performed in human beings with incurable pyloric carcinoma At that time however the realization that the vital processes and their compensation are essentially similar in animals and man had not yet developed sufficiently and operative techniques were not yet advanced enough for the significance of these experiments to be understood and the physiological results applied to man The problem of the best method of closing gastric and intestinal wounds had occupied surgeons for a long time and arose again and again The most prominent French English and German surgeons have studied this question in the present century and after Lembert had discovered the only correct principle for such operations (accurate apposition and union of the serous surfaces) there were increasingly frequent reports of successful intestinal sutures of accidental wounds It was of course a long time before surgeons ventured to excise diseased segments of the intestines New and assured progress in this direction was not made until the last decade In 1871 I pointed out that sections of the oesophagus could be removed in large dogs and that the oesophagus then healed well leaving a slight easily dilatable stricture Czerny was the first to perform this operation successfully in man Then followed Czerny s experiments on excision of the larynx after which a few years ago I successfully removed a human larynx filled with cancerous growths Then came the experiments of Gussenbauer and Al v Winiwatter on the resection of pieces of stomach and intestine which were subsequently confirmed and extended by Czerny and Kaiser Martini s and Gussenbauer s success with resection of the sigmoid and mine with gastrorrhaphy (1877) showed that further progress was possible in this direction The latter operation removed our fears that the scar in the stomach would be digested again by the gastric juice so I concluded my report on this operation with the words There remains only one bold stride between this operation and resection of a carcinomatous segment of the stomach

All this is for the reassurance of those who think that the present operation is a foolhardy experiment on man There is no question of this Gastric resection like any other operation has been fully prepared anatomically physiologically and technically by my pupils and by myself Every surgeon who has any personal experience of these animal experiments and similar operations on man becomes convinced that gastric resection is bound to succeed

This conclusion was also reached by Pean the *Parisian surgeon with the* greatest experience of laparotomies In 1879 he resected 6 cm of *carcinomatous* pylorus of a patient already exhausted by the disease who died on the 4th day after *operation* His operative technique and especially his suture material (catgut) seem to me to have been unfortunately chosen so I cannot attach much importance to this failure The operation does not seem to have encouraged Pean *very much* or he would probably have repeated it which as far as I know he *has not done* nor has any other surgeon to my knowledge ventured to perform this not altogether easy *operation*

The few cases which partly by chance have come my way *in recent years* did not seem to me particularly suitable for a first operation of this kind *Only* last week a woman with an undoubtedly mobile pyloric carcinoma was brought to me by one of my clinical assistants Dr Wolfler After a few days observation and repeated examination I decided on operation with *the patient s consent since* she felt that in view of her increasing exhaustion and inability to keep her food down her end was approaching

This woman of 43 years always pale but formerly healthy and well nourished mother of 8 living children fell ill apparently rather suddenly with vomiting All the symptoms of pyloric cancer soon developed with pyloric stenosis These being well known I shall not repeat *them* Masses resembling coffee grounds were vomited only a few times and the extreme pallor and emaciation of the woman as well as the small rapid pulse had developed only in the last six weeks after the constant vomiting and the small intake of food The only food she could keep down for a time which saved her from starvation was *sour milk*

Preparation for the operation consisted in accustoming her to peptone enemas and gastric irrigation by the usual methods of injection and pumping I shall pass over all the possible difficulties I considered and the operative methods thought of in case the operation should prove impossible to carry out at all or union of *the stomach and duodenum* after the resection should prove impossible and shall reserve the particularly important details of the operative technique for a later more detailed description In view of the patient s great weakness and the probably long duration of the operation (it had taken Pean 2½ hours) I asked one of my experienced private Assistants Dr Barbieri to take charge of the anaesthesia You will understand that it was necessary for me to devote myself to the operation itself with no need to worry about anaesthesia The theatre specially equipped for laparotomies was for well known reasons heated to 24 All my assistants were fully aware of the importance of our undertaking and there was not the slightest disturbance and not a minute of unnecessary conversation

The tumour lying immediately above and slightly to the right seemed to be about as large as an medium sized apple Because of its size it was difficult to expose It proved to be a partly nodular and partly infiltrated carcinoma of the pylorus and over a third of the lower part of the stomach Adhesions with the omentum and transverse colon were divided The small and large omentum were carefully separated Every blood vessel was tied before it was divided The loss of blood was extremely small The entire tumour was moved on to the abdominal wall Incision through the stomach 1 cm beyond the infiltrated part at first only at the back then also through the duodenum An attempt to bring the cut edges together showed that union was possible Six sutures were passed through the

edges of the wound The threads were not yet tied but only used to hold the edges of the wound in position Further incision of the stomach obliquely from above downwards and outwards always 1 cm from the infiltrated part of the stomach Now the oblique wound in the stomach was sutured from below upwards until the opening was only of a size that would fit the duodenum Then the whole tumour was separated from the duodenum 1 cm beyond the infiltration by an incision parallel to that in the stomach (as in an oval amputation) The duodenum was accurately inserted in the opening left in the stomach About 50 stitches with Czerny carbolized silk Cleansing with 2 carbolic acid solution Revision of the whole suture Auxiliary stitches placed at points which seemed weak Replacement in the abdominal cavity Closure of the abdominal wound Dressing

Including the slowly administered anaesthesia the operation lasted 1½ hours No weakness no vomiting no pain after the operation For the first 24 hours nothing but ice was given by mouth Then peptone enemas with wine On the next day the patient was given a tablespoonful of sour milk at first every hour then every half hour The patient a very sensible woman feels quite well lies unusually quietly and sleeps most of the night with the help of a small injection of morphine No wound pain moderate febrile reaction The dressing is still untouched After some trials of broth which the patient did not enjoy feeding continues with sour milk only of which the patient takes about 1 litre a day The enemas of peptone and pancreas easily cause flatulence and colic and have therefore been stopped A rectal injection of a little wine 2 3 times daily is agreeable to the patient Yellowish pulpy stools as in infants The pulse is much quieter and fuller than before operation This has continued up to now without the slightest complication To prove how well she feels I may add that I had to have her moved the day before yesterday to a large ward at her own urgent request because she found too little entertainment in the isolation ward which she shared with a woman also bored who had undergone ovariotomy on the same day

The resected specimen measures (*horribile dictu!*) 14 cm along the greater curvature The pylorus hardly admits a quill The shape of the stomach is not much altered by the operation but is merely smaller than before

I am myself pleasantly surprised by the very easy postoperative course I should have expected more local and general reaction—I might almost say more bad behaviour on the part of the stomach I still hardly dare to expect that everything will go on equally quietly A relapse to the previous state of weakness might still occur This would be the most fatal complication because nothing could be done against it After these six days without reaction the wound and everything around it must be nearly healed so that even if suppuration at some suture or other should occur sudden peritonitis is hardly to be expected However circumscribed suppuration or abscesses might develop around the scar it is to be hoped that we may discover them soon enough to drain them to the outside

The course so far is already sufficient proof that the operation is possible Our next care and the subject of our next studies must be to determine the indications and to develop the technique to suit all kinds of cases I hope we have taken another good step forward towards curing unfortunate people hitherto regarded as incurable or if there should be recurrences of cancer at least alleviating their suffering for a time and I am sure you will forgive me if I take some pride in the fact that it is the work of my pupils which has made this progress possible

Nunquam retrorsum was the favourite motto of my teacher Bernhar Langenbeck it must also be mine and my pupils

WOLFLER A (1881) Gastro Enterostomie (Gastro enterostomy) *Zbl Ch* 705 708 (Nov 12)

This is the name I would give to an operation I carried out on Sept this year on a patient with inoperable pyloric carcinoma

Through the kindness of Dr Kauder Assistant at Dr Bamberger s C *a 38 year old man Michael Gold who had suffered from symptoms of canc* the stomach for six months was brought to Prof Billroth s Clinic on Sept

The patient was very weak and emaciated *as he had vomited most o* food for the last 3 months Recently he had been able to take only a quantity of fluid provided the considerably distended stomach was washed once a day

On examination under chloroform anaesthesia a tumour the size of a h *was felt in the pyloric region The fact that it was freely movable in all direc* made me decide on an exploratory incision

After opening the abdominal cavity I noticed that although the pyloric c noma was still freely mobile it had infiltrated the hepato duodenal ligament the head of the pancreas In the circumstances it seemed that resection of pylorus was possible but with no hope of complete extirpation of the tumour

If the abdomen was not to be closed without achieving anything the ma *of a nutritional fistula in the intestine had to be considered*

The disadvantages of such an operation are obvious (1) If the fistula no longer be made in the accessible upper part of the duodenum the impor biliary and pancreatic secretions are excluded and (2) feeding through a fis must be a very distressing condition for any patient

I therefore decided to make a direct communication between the stomach the small intestine and did this by the following method I opened the stom *a finger s breadth above the insertion of the gastro colic ligament* on the gre *curvature making a longitudinal incision 5 cm long* Then I brought up a l of small intestine made a slit of the same length in it on the side opposite mesentery and inserted the edges of the intestinal opening in the gastric open by joining the posterior edge of the intestinal slit to the posterior edge of *gastric slit with silk thread by means of the interior ring sutures* described me At the anterior edges of the gastric and intestinal slits the mucous membra were first sutured and then the serous and muscular layers brought together means of modified Lembert sutures *

After careful disinfection of the whole field of operation with carbolic ac the abdominal wall was sutured *and the closed wound covered* with an iodofo dressing Throughout the operation the stomach and the loop of intestine to attached to it lay on sterile sponges

* See the technique for these intestinal sutures in WOLFLER (1881) *Uber die von Her Prof Billroth ausgefuhrten Resektionen des Karcinomatosen Pylorus* Wein p 23 Pl 1 figs 10 11

To avoid the escape of *intestinal contents* I loosely tied the afferent and efferent parts with thick silk thread passing through the mesentery This seems to me the simplest type of temporary occlusion as recommended by Schede

The operation was carried out with strict antiseptic precautions but without the spray No drainage of the peritoneal cavity The post operative course was satisfactory in every way The patient remained completely afebrile and felt steadily better from the day of operation onwards The distressing vomiting stopped and the patient was able to take increasing quantities of food liquid at first and solid after the 8th day without causing any harm The abdominal wound healed by first intention under a single dressing

The patient is now in the fourth week after operation and has daily evacuations of firm brown stools

By means of gastro-enterostomy I have excluded the carcinomatous pylorus from the alimentary pathway and have provided a new route for food arriving in the stomach without at the same time excluding the passage of the bile and pancreatic secretion by way of the afferent loop The many chiefly physiological questions which arise in this connection can probably not be answered until later This much is certain however *any theoretical objections* which can be made to this operation are invalidated by the *results obtained* Further experience may show us that this type of plastic operation may enable us to avoid an intestinal fistula not only in gastric but also in intestinal carcinoma

On Oct 2 this year Prof Billroth carried out the same operation in a man 45 years old named Georg Stadler who also suffered from an inoperable pyloric carcinoma which as could be seen after laparotomy had already spread far up the lesser curvature and towards the head of the pancreas The operation went quite smoothly and was completed in 1 hour No symptoms of peritonitis developed afterwards The intestinal canal remained patent because the patient did occasionally pass stools However vomiting of bile set in on the day after the operation and continued until the patient s death 10 days after the operation Post mortem examination showed no peritonitis The inserted intestinal loop which was only 11 cm from the duodenum was solidly fused with the edges of the gastric opening The reason for the vomiting of bile was that when the loop of intestine was pulled up a *spur* was formed which divided the gastro intestinal lumen 4 cm in diameter into two *unequal* parts the *larger* part belonged to the loop *bringing in* the bile and pancreatic fluid and the *smaller* belonged to the *efferent* loop of the intestine and lay hidden under the somewhat overhanging edge of the gastric opening The bile and pancreatic fluid had therefore passed into the stomach instead of into the efferent loop

This instructive finding shows that in future cases we must see to it (1) that we choose a loop in which we know which side *brings in* the bile and pancreatic fluid and (2) that we attach the loop in such a way that the part of the lumen belonging to the afferent loop is mostly covered by *intact* gastric wall this is probably best done by attaching it to the stomach by a few stitches The efferent loop on the other hand must communicate with the gastric cavity through an opening 2 3 cm in diameter

That satisfactory nourishment can be provided by gastro enterostomy is proved by the result obtained in the first case

Vienna late October 1881

HACKER VON (1885) Zur Casuistik und Statistik der Magenresectionen und Gastro enterostomien (Case reports and statistics of gastric resection and gastro enterostomy) *Verh dtsch Ges Chir* **14**, *pt* 2 62 71

In his lectures Dr Billroth has often expressed the idea that pyloric carcinomata which *extend far up the lesser and greater curvatures* so that the duodenum and stomach cannot be brought together after sufficiently wide resection or the union cannot be made without danger of tension on the sutures might be dealt with by removing the pathological tissue regardless of the possibility of union closing the stomach and duodenum and making a communication between the stomach and *jejunum by means of a Wolfler gastro enterostomy In this way one might remove* carcinomata confined to the stomach with some hope of effecting a radical cure Even if it were not possible to remove all the diseased tissue there would still be the advantage that the degenerating carcinoma would be isolated the source of haemorrhage thus eliminated and the addition of cancerous juices to the gastric juice avoided The chances of prolonged *well being after the operation* would therefore be much better than after simple gastroenterostomy

This plan was put into practice on Jan 15th this year The patient was a *man of 48 years who had all the symptoms of a stenosing pyloric carcinoma* When the abdomen was opened the tumour the size of a fist very mobile and non adherent could be drawn forward and it was seen that it reached far up especially along the greater curvature As the patient was in very poor condition Prof Billroth first performed a gastroenterostomy so that if dangerous symptoms set in the operation could be stopped at this point The gastroenterostomy was carried out by Wolfler s method The jejunum was pulled up over the transverse colon and fixed to the stomach in front of it Since the patient was not collapsed after this rapidly executed operation and the pulse was strong extirpation was immediately undertaken The tumour isolated in the usual way from the greater and lesser curvatures was first separated from the duodenum which was held compressed by *an Assistant s hand The duodenum was then turned in to its lumen* and closed with Lembert sutures in two layers When the cavity of the stomach was opened it was immediately cleaned out with a separate sponge and another was pushed inside the part to be resected Then followed its separation step by step from the healthy part with immediate application of occluding sutures with a few Lembert sutures in the intervals Closure of the stomach was also carried out with the help of manual compression In the course of excision it was found that the cut downwards on the greater curvature had to be taken to within ¼ cm *of the previously made gastro intestinal fistula* because of the extent of the carcinoma and even there the cut surface was still infiltrated The two operations including closure of the abdomen lasted 1¾ hour The subsequent course was entirely *favourable and free from reaction The patient was given fluid nourish* ment on the next day and light meat after a week Vomiting had stopped and recurred only once after an over large meal but as the patient began to eat more marked meteorism and obstinate constipation developed which could be overcome only with daily enemas and laxatives or daily Hegar s irrigations It was doubtful whether these symptoms were due to the rapid passage of food into the intestine without actual gastric digestion or as seemed more likely in this case to compres sion of the transverse colon by the jejunum as in Lauenstein s case Four weeks

after the operation the patient left the hospital but has continued daily enemas to obtain bowel movements Recently 14 months after operation gastric symptoms have reappeared with vomiting on one occasion and an induration can be felt in the gastric region so it seems likely that a recurrence is developing from the point mentioned as being infiltrated

Although it has therefore not been possible to achieve a radical cure in this case the practicability of the operation has been demonstrated In similar cases in future if gastro enterostomy is to be performed first as a precaution the fistula should be made still further to the left towards the fundus It would however be technically easier to carry out the resection first The lower part of the incision in the stomach could then be used directly for the insertion of the jejunum especially if owing to extension of the carcinoma on the greater curvature the incision there were made curved

In Prof Billroth s hospital and private practice resection of the pylorus has been performed 18 times altogether including the present case—8 times with good results Since not all the cases have been published and only a few of those not published have become known I should like in the interests of accurate statistics to add here a short review of them and refer the reader for details to a compilation of all the gastric operations carried out in this Clinic since 1880 which is to be published shortly

In 15 cases the operation was performed for carcinoma (with 7 successes) in 3 cases for cicatricial stricture (with 1 cure) In 2 cases the operation was not the typical circular resection of the pylorus One of these was the case just described in which the duodenum and stomach were closed separately and a gastroenterostomy was performed The second was in a woman of 47 years in whom a partial pyloric resection was performed on May 3 1883 because of a cicatricial stricture following an ulcer and severe cicatricial contraction in the lesser curvature A wedge with its base towards the lesser curvature was removed without interruption of the continuity of the stomach and duodenum along the greater curvature Death from collapse 12 hours after operation Post mortem examination of this woman who was in very poor condition with severe kyphoscoliosis revealed advanced atrophy of all the internal organs

In 16 cases typical circular resection of the pylorus was carried out 7 times with good results in two cases (with 1 success) for cicatricial stricture One of these cases (man 43 years old operation 3 viii 1882 pyloric stenosis from nitric acid poisoning normal postoperative course death on 7th day from suppuration of sutures) has been briefly mentioned by Czerny (*Wien med Wschr* 1884)

The 2nd case (in a man 31 years old) operation 3 v 1884 cicatricial stenosis probably following an ulcer with kinking of the pylorus by a band running to the liver) has not been published before Here the operation was very difficult It had to be performed deep under the right costal arch under the raised liver The duodenum was extremely thin walled adhesions with the pancreas had to be divided and the pancreatico duodenal artery ligated In spite of this the post operative course was free from complications The patient who had been very weak and unfit for work recovered quickly and can presumably be regarded as radically cured His appearance now a year after the operation is flourishing

In 14 cases typical resection was carried out for carcinoma 8 times with good results Since adhesions are of the greatest importance in determining not only

the immediate but also and more especially the final result in pyloric carcinoma I shall place the cases in 3 classes according to whether there were no adhesions slight easily loosened adhesions (e g with the lesser or greater omentum or with isolated removable glands) or lastly extensive adhesions (especially with the liver pancreas etc)

Of these 14 cases only 2 were in the first class These are Wolfler's well known case (52 year old woman Gebhardter operated on on 8 ii 1881) and a case operated on by Dr Billroth (46 year old woman Schuhmann operated on on 24 v 1883 see *Wein med Wschr* 1883 no 41) In the latter case the period of observation is now 1½ year and so far the patient feel perfectly well and there is no trace of recurrence In the woman operated on by Dr Wolfler it is well known that a small carcinomatous nodule was removed from the scar on the abdominal wall on July 5 1883 (see *Wein med Wschr* 1883 no 41) It is interesting that this nodule did not reach down to the inner surface of the peritoneum so one can here suppose that the cancer arose from inoculation during operation as in the cases recently collected by Kraske (see *Zbl Chir* 1884 no 48) In autumn 1884 when the patient appeared again there was a swelling the size of a fist firmly attached to the iliac crest and a glandular swelling over Poupart's ligament in the iliac fossa Surprisingly the swelling became considerably smaller in the course of a few months under the influence of rest and moist heat There is not the slightest hardening to be felt in the stomach itself and digestion is perfectly normal

Of the 7 cases in the second category 3 died after operation (collapse peritonitis starvation) and 4 ran a favourable course Three of these have since died (2 from recurrences after 4 and 10 months respectively and one after gastroenterostomy performed after a year because of a recurrence)

In the 4th case a woman 37 years old operation July 16 1884 by Prof Billroth) a recurrence has been developing according to a letter since January 1885 (6 months after the operation)

The 5 cases in the 3rd category all died from collapse or peritonitis immediately after the operation (after 12 to 31 hours or during the first few days)

After this review I am inclined to believe that in cases in the first and second class a cure or at least a considerable prolongation of life can be achieved but that cases in the 3rd class should in future be excluded from this operation For these cases gastroenterostomy would be appropriate if there were signs of stenosis The reported results of gastric resection may be regarded as fairly good It must be remembered that in the period when this operation was being tested it was used in cases which we now exclude and one must not demand more from surgical treatment in pyloric carcinoma than in any other carcinoma The single case in which there is no recurrence now four years after the operation surely gives clear evidence that at least a considerable prolongation of life can be achieved by operation which is even now denied by some not only for gastric but also for most other carcinomata It is also to be expected that the results will improve still further in the future when the problems of indications and technique are well on the way to solution Our own results have considerably improved as we lost 8 of the first 12 cases but only 2 of the last

I venture also to show you two specimens to illustrate the way in which the jejunum was joined to the stomach in a gastroenterostomy which I performed as deputy for Prof Billroth on March 22 this year

The jejunum in this case was brought into contact with the posterior wall of the stomach through a slit in the transverse mesocolon without transverse rupture of the gastro colic ligament or of the mesocolon For this reason and because death was not related to the operation the case is not without interest In most cases as is well known the jejunum is drawn forward over the transverse colon laid against the anterior wall of the stomach and fixed there Now there are no known cases in which this method of insertion has produced conditions which might lead to obstruction but compression of the transverse colon by the jejunum was observed in a case of Lauenstein s which however only led to delayed evacua tion and in our case in which gastroenterostomy preceded resection of the pylorus and obstinate constipation exists such a compression is very probable I cannot help thinking that the jejunum pulled over the transverse colon might still one of these days cause serious disturbances of the passage through the intestines If a higher part of the jejunum is inserted in the stomach it may compress the transverse colon like a stretched band If a part far below the flexure is chosen intestines with a long mesentery might become enclosed in this loop and if this is not greatly to be feared because of the rapid growth of adhesions something would have to be done to hinder the passage of gastric contents into the afferent loop In one of our cases dating from the time when we still used any loop of the intestine which presented itself it is known that intractable vomiting and death from starva tion occurred because the gastric contents ran back into the afferent loop Wolfler then set out instructions which made it easy to find the uppermost loop of the jejunum and also showed how to insert the loop situated about 40 cm from the flexure so as to make the afferent loop narrower and to bring the efferent loop into more direct contact with the stomach by a kind of valve This last would be difficult to achieve with certainty in practice or at least would make the operation more complicated If the point at the right distance below the flexure is found the jejunum coiled round the transverse colon will be so stretched when the colon is distended that the opening into the efferent loop will become free of its own accord In view of the doubts expressed about raising the jejunum I made experi ments on cadavers to determine the most natural method of joining the jejunum to the stomach It is clear that it is also possible to reach behind the transverse colon through the transverse mesocolon to the stomach (the posterior wall) Courvoisier has done this in one case but cut extensively through the gastrocolic ligament and detached the mesocolon from the transverse colon for a considerable distance I should not care to recommend this method of union Lauenstein rightly objected to this method on the ground that it is not only more complicated but also according to our present experience carries with it a risk of gangrene of the transverse colon When one examines the mesocolon of a large number of cadavers one finds provided there is no inflammatory thickening and the subject is not obese (a condition not likely to be encountered in cases of stenosis where gastroenterostomy is indicated) that there are usually some remarkably thin parts whose transparency shows them to be almost avascular—parts round which the vessels supplying the mesocolon (branches of the right middle and left colic arteries) pass round in a wide arc In these parts one can easily make a slit without doing any damage

wide enough for the jejunum to be fixed through it to the posterior wall of the stomach I have not enough experience to say whether this can be done in every case

I have carried out a gastroenterostomy by the method I have suggested in a farmer 49 years old who had symptoms of pyloric stenosis and intense gastric pain He was very weak and cachectic He was absolutely convinced that he had an animal in his stomach whose egg he had swallowed with some dirty water He daily demanded operation believing this to be his only hope of recovery There was no definite tumour but only an indistinct tender firmness below the right costal border After opening the abdomen which allowed a considerable amount of fluid to escape a pyloric tumour the size of a fist could be felt fixed at the back not easily movable and impossible to pull up into the incision on the linea alba Resection being impossible I set out to do a gastroenterostomy The omentum and transverse colon were turned up The stomach was pushed downwards and I made a slit in the mesocolon in the manner described parallel to the course of the vessels and widened it with scissors without causing any haemorrhage To prevent contraction of the slit in the mesocolon and consequent compression of the jejunum I fixed its edges to the stomach by a few (6) superficial sutures so that it surrounded a circular area of the posterior wall of the stomach I chose the part of the jejunum below the duodenojejunal flexure for insertion because I had learned from experiments on cadavers that when this part was raised the afferent segment falling in the curvature developed a slight kink of its own accord The loop of jejunum (after milking out its contents) was kept closed by 2 thick silk threads pulled through the mesentery and lightly tied The escape of gastric contents was effectively prevented by an assistant s hands When openings of 5 6 cm had been made in the stomach and in the gut their lower edges were joined together with special accuracy since these sutures could not be re examined afterwards making internal intestinal sutures with mucous membrane sutures between them Then followed the external intestinal sutures and Lembert s sutures The site of implantation was everywhere at least ½ cm from the border of the slit in the mesocolon The operation lasted no longer than in the simpler cases when the other method was used The patient died 36 hours after operation from collapse I can therefore say nothing about the result of this method of implanting the jejunum from observation of the subsequent course It was found after death however that when water was poured into the stomach most of it flowed in a stream through the efferent loop and very little through the afferent kinked loop of the jejunum Post mortem examination (Dr Zemann) showed severe anaemia and sero purulent peritonitis The wounds in the abdominal wall and the gastrointestinal fistula were well approximated and closely adherent The intestinal loop concerned showed no changes but the extensive carcinoma of the pylorus shows extensive splitting up in the inner parts and on the anterior wall this destruction reached close to the serous layer which shows marked reddening and partial discoloration at this point

The peritonitis obviously arose from the carcinoma which was on the point of breaking through to the peritoneum and peritonitis had already set in at the time of operation From all this I gained the impression that the operation can be performed by this method that it is no more complicated than any other since there is no need to divide the gastro-colic ligament or to detach the mesocolon and finally that insertion of the jejunum by this method is anatomically natural and

does not require any special measures to constrict the afferent loop which becomes kinked naturally On the other hand I cannot deny that it may be objected that the operation is carried out to a greater extent in the abdominal cavity itself and that the parts of the intestine concerned are not drawn forward and united as it were extraperitoneally as in Wolfler s method so that it is particularly neces sary to protect the surrounding viscera during operation It is also more difficult to hold the stomach closed by means of an assistant s hands I think however that this method of insertion should be considered if it is found that compression of the transverse colon tends to occur when the jejunum is laid over it and that this may cause serious complications *and also in cases in which the disease has spread too far towards the fundus on the anterior wall of the stomach and the posterior wall has remained free*

There remain only a few more words to be said about the numbers and results of our gastroenterostomies *So far the operation has been performed 9 times in Billroth s Clinic with 5 deaths and 4 improvements In one case there* was as was discovered later a tuberculous stricture of the duodenum and upper jejunum The improvement was short lived Two months after the operation the patient died from wasting after aggravation of the pulmonary and intestinal tuber culosis In 8 cases there was carcinoma of the pylorus In 5 of them death followed the operation and in 3 some improvement was obtained In one of these (Wolfler) death from wasting occurred after a month in a patient who was also suffering from tuberculosis Two were operated on 2 and 1½ months ago respectively One of these is the case in which pyloric resection was carried out after gastro enterostomy The other is that of a 48 year old woman Here a fairly long period of well being may be expected because digestion has become perfectly normal and the improvement in general condition is remarkable

Speaking generally the results of this operation are not very good This is understandable since the most usual indication is inoperable carcinoma in other words the patients were often already too cachectic to stand such an operation or to recover after it In carcinoma as a matter of fact the operation does not aim very high it is only expected to prolong life for a short time free from the distress caused by stenosis This end was achieved in some of our cases and once each by Lucke and Socin In Socin s case the general condition was good 4 months after operation Rydygier has performed the operation in 2 cases of ulcer with stenosis with good results What Wolfler said about this operation has therefore proved true By this means it will be possible to relieve carcinoma of the pylorus and to cure cicatricial strictures

POLYA E (1911) Zur Stumpfversorgung nach Magenresektion (Method of dealing with the stump after gastric resection) *Zbl Chir* 38, 892 894 (No 26)

Of the various methods which have been suggested for dealing with the stumps of the stomach and duodenum after resection of the stomach it is undoubtedly *Kocher s that produces* the most nearly normal anatomical relations but after extensive resection even if the duodenum can be mobilized gastroduodenostomy cannot be carried out without tension and this easily leads to failure of the sutures Moreover if the stump of the stomach is too small and the opening for the

anastomosis in its posterior wall has to be made too close to the suture closing the stomach the intervening strip of stomach wall may well die as has been seen more than once Kocher does remark quite rightly that in cases where the implantation of the duodenum cannot be made secure the suture closing the duodenum will not hold securely either but insufficiency of the gastro duodenal anastomosis does much more harm than insufficiency of the suture closing the duodenum which with sufficient plugging leads at most to a fistula which will close sooner or later The other methods used are decidedly inferior to Kocher s In Billroth I the danger of insufficiency of the sutures is even greater In Billroth II there is a risk of regurgitation especially if the gastric stump is too small and prevents us from making a suitably placed gastroenterostomy of appropriate size The method of Kronlein Henle and Mikulicz combines the disadvantages of Billroth I and II and Rydygier s procedure is rather complicated and slow The method I should recommend but only in extensive resections where Kocher s method of union cannot be carried out easily and without tension consists essentially in end to side implantation of the whole stump of the stomach in the uppermost loop of the jejunum drawn through a slit in the mesocolon In this way

1 The gastrojejunostomy is easily performed
2 The gastric stump can be joined to the intestine without any tension at all
3 The suture holds perfectly securely because wide areas of serous membrane can be laid together everywhere
4 Conditions are as favourable as possible for emptying the stomach because (a) the communicating opening is very large (b) the opening at the distal end of the gastric stump lies in the direction most nearly corresponding to the physiological evacuation of the stomach and (c) the stomach opens into the upper part of the jejunum and there is therefore no afferent loop as in Mayo s no loop gastro enterostomy which I used as a model in devising the opening into the intestine
5 We avoid making the closing suture of the stomach

I prefer Kocher s method for the less extensive resections in spite of these advantages and the radiological evidence that the gastric stump is evacuated more quickly chiefly because with his method the field of operation is entirely limited to the upper part of the abdomen the part between the lower surface of the liver and the transverse mesocolon On the other hand I think the present method might be used to advantage for exclusion of the pylorus

I have so far used this method in six cases of extensive resection of the stomach for carcinoma Two patients left the hospital cured a third a woman of 70 years was perfectly well for 5 weeks and was kept in hospital only because of a small duodenal fistula Then erysipelas of the nose developed and spread over the whole head finally the small abdominal fistula became infected and the patient died 49 days after the operation from an acute retroperitoneal phlegmon Three patients in very poor condition (one extremely anaemic and two with lymph gland metastases) died 1 2 days after operation but in these as post mortem examination proved the gastro jejunal anastomosis held perfectly X ray examination in two cases showed that bismuth paste left the stomach in a very short time

The operation is greatly facilitated if the serous suture of the jejunum to the posterior wall of the stomach is made before the stomach is amputated when it has been completely liberated and cut off from the duodenum Then the stomach

is removed the jejunum opened at a suitable point and the gastro jejunostomy completed by a circular through and through suture and an anterior suture of the serosa

FINSTERER H (1918) Ausgedehnte Magenresektion bei Ulcus duodeni statt der einfachen Duodenalresektion bzw Pylorusausschaltung (Extensive resection of the stomach in duodenal ulcer instead of simple duodenal resection or exclusion of the pylorus) *Zbl Chir* 45 434 435 (No 26)

The fact that after simple gastroenterostomy or von Eiselberg s unilateral exclusion of the pylorus for duodenal ulcer *peptic ulceration* may occur at the point of anastomosis led me to *remove at least a half or two thirds of the stomach* in *all cases of resection of the duodenum or exclusion for duodenal ulcer* starting from the idea that a decrease of the secretory area of the stomach would effectively combat the hyperacidity

In the last 2½ years I have carried out extensive gastric resections in 24 cases of duodenal ulcer operated on at the Garrison Hospital In 19 of them the part of the duodenum bearing the ulcer was also removed and in 5 the operation was done to exclude the ulcer which was left If the ulcer is in the upper horizontal segment it is possible to *resect the duodenum* and I have always done so (19 cases) If the ulcer is further out and if it reaches to the neighbourhood of the papilla it is better not to extirpate it because of the difficulty in dealing with the duodenal stump but to *exclude it* by removing the beginning of the duodenum together with the pylorus and half the stomach in which case the duodenal stump is easily dealt with above the ulcer If however the ulcer is so large that it reaches from the pylorus to the papilla the cut must be made in the stomach itself immediately beside the pylorus the small gastric stump being closed after excision of as much as possible of the mucous membrane In all cases the stomach and intestine are joined by the end to side method which I have found very satisfactory in more than 200 resections

The extension of the resection to half the stomach does not appreciably extend the operation because it is only the removal of the duodenum which causes difficulties and not the extirpation of the freely movable segment of the stomach Among my 24 patients there was only one death in a patient with *very severe duodenal haemorrhage* who was *almost completely exsanguinated* at the time of operation without palpable radial pulse and died from acute anaemia All the other patients recovered without complications

In *duodenal resection* which Clairmont and von Haberer have already performed in a very large number of cases *the pre pyloric part of the stomach which is removed at the same time amounts to no more than a very narrow strip* whereas in my cases the length of the part removed is at least 10 15 cm along the lesser curvature and 20 30 cm or more along the greater Schnitzler has argued theoretically in favour of extensive resection in *gastric ulcer* if only because of the possibility of multiple ulcers but in practice as far as can be made out from his lecture he has carried out only one subtotal gastrectomy for peptic ulcer Hofmeister also seems to prefer pyloric resection to transverse gastric resection for gastric ulcer

After such extensive gastric resections the patients no longer have the slightest symptoms of hyperacidity and can therefore feed perfectly normally in a very short time The scientific explanation of the disappearance of hyperacidity is given by Edkin s experimental studies Whether it is possible to cure hyperacidity *permanently* by resecting half the stomach can only be decided after many years observation but since the operation is *no more complicated* than von Eiselsberg s simple exclusion of the pylorus and since the *slight prolongation of the operation* caused by tying the gastro colic ligament and the lesser omentum is no longer of any importance since *local anaesthesia* has been used on principle for all gastric operations I would recommend *removal of the greater part of the stomach* (up to ⅔) *in the treatment of duodenal ulcer both when resecting the duodenum and when excluding the ulcer* in order to save the patient from the danger of a recurrence of the ulcer and from peptic ulcer of the jejunum If such a proposal has ever been made elsewhere I can only support it in full

SCHOEMAKER J (1922) Zur Technik der Magenresektion nach Billroth I (Contribution to the technique of gastric resection according to Billroth I) *Arch klin Chir* **121**, 268 271

The simpler the conditions I leave after an operation the better I am pleased For this reason I always make an end to-end anastomosis after resection of the large intestine and always use the Billroth I method for resection of the stomach It has been said that after extensive resections it is sometimes difficult to bring the stomach and the duodenum together without tension but this is not true if a kind of tube is made out of the fundus of the stomach such as I described in *Arch klin Chir* in 1910 For this the stomach must not be cut straight across but along the line shown in fig 43 The technique of suturing however was not clean enough to satisfy me so I have changed it

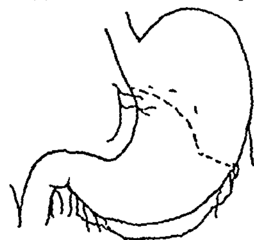


FIG 43
Schoemaker s operation line of division of the stomach

The procedure for resection of the stomach is now as follows I begin with the duodenum set it free by tying the greater and lesser omentum and close it with one of the small clamps for large intestine which I have described here before A heavier clamp is put on the stomach because I must be able to pull on it A cut is then made immediately alongside the duodenal clamp so that no mucous membrane remains on this side of the clamp On the stomach side however a strip of about 1 cm is left outside the clamp which would otherwise slip off when pulled This strip however must be covered so a piece of metal which I call a protector is placed over the clamp

The stomach is now pulled up and freed from its attachments and adhesions For cancer I follow the lesser curvature as far as I can that is almost to the cardia

for peptic ulcers I go only far enough to reach above the infiltrated zone Along the greater curvature I go to the point where my gastric tube is to end When this has been done the stomach must be closed along the line shown To the short straight piece at right angles to the greater curvature I apply another small colon clamp and for the curved part I use a special instrument This is intended to fulfil two conditions Firstly it must hold the stomach so firmly that the mucosa disappears but the serous membrane cannot slip out even if I cut close along the instrument secondly I must have a free strip of serous membrane so that I can suture it even if I leave the instrument in position This is why it is double

The clamp is now placed in position as shown in fig 44 and screwed up very firmly

Then the part of the stomach which is to be removed is closed by two ordinary clamps and cut off close against the large and small instruments

When I now remove the outer half of the clamp I have a small strip of free serous membrane which I can very easily stitch over with a continuous catgut suture When this suture is complete I take off the second half of the instrument and make a continuous silk suture of the serosa over the first one This completes the tube of stomach

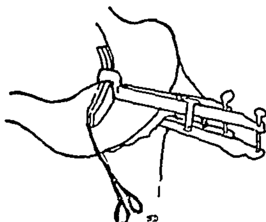


FIG 44
Schoemaker s operation application of the large double clamp and of the small clamp on the greater curvature side

This now has to be joined to the duodenum which is always easily done because the distance from the cardia to the duodenum in a straight line is always shorter than the length of the tube of stomach when pulled straight

The two colon clamps are therefore brought together and the serous membrane at the back is joined with button sutures Ordinary intestinal clamps are now placed on the duodenum and on the tube of stomach and the smaller clamps are removed

A circular catgut suture through all the layers and a continuous silk suture through the serous membrane on the anterior wall complete the operation

If there is any anxiety about the point when three sutures meet a flap of serous membrane can be drawn from the stomach to cover it but this is not essential

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